INTRODUTCTION

Ketosis is a condition of formation of ketone bodies (aceto acetic acid, beta hydroxyl butyric acid and acetone) and measures the level of capacity to convert circulating non esterifies fatty acid into glucose via gluconeogenesis process. This condition is seen just after pregnancy or prior calving. Low body condition score (BCS), low feed quality and hepatic insufficiency are the pre disposing factor of ketosis (Fincher *et al.*1995). Animal show low production, weight loss, ruminal dysfunction that result the great economic loss of a farm. Two different types of ketosis that is based on clinical appeared or not. Diagnosis of ketosis based on analysis of ration, periodic blood tests, BCS, which can be easily applicable in dairy farm(Boddie,1995). In dairy cattle there is a time range when animal affected by various metabolic and infectious diseases like milk fever, retain placenta metritis and mastitis which are associated with alteration of diet or loss of immunity (Drackley *et al* 2005). Among these one major clinical condition is ketosis. It is associated with intense milk production and negative energy balance which occurs in most productive stage of lactation especially in early lactation results in negative energy balance due to failure to provide sufficient glucose when the animals are subjected to heavier demands on their resource of glucose and glycogen (Esposito, 2014). Dysfunction of adrenal gland, stress of parturition, lactation and stress of malnutrition leads to decreased adrecocorticotropic hormone (ACTH) activity. Moreover types of rations are also responsible for ketosis. Ensilage is more ketogenic than hay. Animal 4-10 weeks posrt partum period, peak milk production period, decrease dry matter in food are prone to ketosis. Again starvation also leads ketosis as propionic acid decrease resulting utilization of fat. From qualitative and quantitative estimation of ketone body in rumen liquor it appears that abnormal ruminal condition play an important role in the production of clinical ketosis (Foster, 1988)

When feed intake does not fulfill demand of energy balance that cause insufficient production of propionic acid. For this reason enough glucose could not formed that results low blood sugar. For that fatty acid and glycerol oxidise to form acetyle co A. When produce excess acetyle co A, which convert into ketone body and results ketosis (Holmes, 1992)

CASE HISTORY, CLINICAL SIGNS AND DIAGNOSIS

An owner of a cow came to a veterinary practitioner in CVASU complaining that one of his cross breed Holstein Friesian cow having body weight 350 kg, age about 3.5 years losing body weight gradually. As per the owner complaint the milk yield was drastically decreased from 7 liters to 2 liters. Body condition score was very poor that was three in the scale. The owner showed the treatment history to him. After hearing everything he sent me as an intern doctor for physical examination of that cow.

Physical examination showed that ruminal movement was lower than normal (three times in two minutes). The urine was red in color. Feces was semisolid, no visual worm in the feces. Others physical parameters like heart rate (65/min), respiratory rate (25/min), temperature (102 F) were in normal condition. Ruminal pH (6.5) was normal in state. Mucous membrane was pale in color.

After physical examination the doctor advised to do some laboratory tests including complete blood count, some serum biochemical examination (serum glucose, triglycerides, calcium, magnesium, total protein, and serum glutamic oxaloacetic transaminase), routine examination of urine, feces and blood protozoa. Following standard protocol samples were collected for performing the tests.

Fecal examination: This test was performed by sedimentation method. For this the sample was taken into a test tube containing sedimentation fluid. Keep the tube for thirty minutes. Then sediment was collected and placed it in a slide and observed it under microscope (Ritchie, 1948)

Blood protozoa: a smear was made on the slide and air dry. Place the slide in 1N HCL solution. Rinsed the slide with water and flood with Giemsa stain for five minutes. Water wash the slide and observed under oil immersion (Goto K et al. 1978).

No blood protozoa was found.

Table 1: Blood examination (Complete Blood Count)

For blood examination blood was collected from jugular vein and keep into a vacutainer without no anticoagulant. Then the sample was kept about one hour. Blood cell settled down and supernatant clear fluid (serum) was collected. Then it was centrifuged at 2000 rpm for 15 minutes. Then supernatant was collected. This clear serum was insert into auto blood analyzer machine (Humalyser 3000) following the protocol.

Parameters	Result	Normal range
Hemoglobin	9.8	8-15gm%
Erythrocyte sedimentation rate	0	0-1(mm in 1st hour)
Total Erythrocyte Count	6.8	5-10miilion/cumm
Total Leukocyte Count	9	4-12thousand/cumm
Packed cell volume	33	24-46%
Differential Leukocyte Count		
Lymphocytes	50	45-75%
Neutrophils	45	15-75%
Eosinophil	5	0-20%

The hematological parameters were found within the normal limit. Normal level of neutrophils, lymphocyte, and eosinophil indicates that there is no inflammatory reaction is going on in the body.

Table 2: Routine Examination Urine:

Parameters	Test Result
Color	Reddish
pH	7.2
Specific gravity	1.03
Blood	++
Ketone	++
Protein	-
Creatinine	100mg/dl
Nitrate	-
Bilirubin	+
Leukocyte	+

In the table no 2 we can see that the pH of the urine, creatinine level specific gravity are normal limit but there was presence of Red blood cell and Ketone bodies in the sample. Red blood cell indicates that there may be abnormalities in kidney like nephritis or glomerulonephritis. Ketone bodies may present due to ketosis in the cow.

Table 3: Serum Biochemistry Analysis

Parameters	Result	Normal Range
Calcium	8.6	9.7-12.4mg/dl
Magnesium	1.6	1.8-2.3mg/dl
Phosphorous	2	5.6-6.5mg/dl
GOT(AST)	59.1	78-132 U/L
GPT	28.8	11-40 IU/L
Glucose	40	45-75mg/dl
Triglyceride	62.3	0-16mg/dl
Total protein	63.2	67-74 g/l
Creatinine	1.5	1-2.6 mg/dl
Calcium :Phosphorus	4:1	2:1

In the table no 3 we can see that the limit of serum calcium, magnesium, glucose, serum glutamic oxaloacetic transaminase is lower than the normal range that indicate mineral deficiency, hypoglycemia and liver damage. On the other hand serum triglycerides is higher than the normal value that indicate negative energy balance (Grummer, 1993).

Therefore over all we see that the level of calcium magnesium and phosphorus found lower than normal range that due to lack of mineral in feed as the cow is lactating so huge mineral pass out with milk. Serum glucose found lower whereas triglycerides levels found higher than normal level, which indicate negative energy balance and presence of ketone bodies in urine suggests keto acidosis. The above clinical alteration suggests that the cow was suffering from clinical ketosis (Boisclair *et al*, 1987)

Treatment that was given includes intravenous infusion of 1000ml 10% dextrose solution daily for 3 days. Combine preparation of calcium phosphorus and vitamin D (Inj. Cal-D-Mag) was given intravenously 200cc daily for three days. A kidney tonic (sol. Nephrocare) 10 Tea spoon twice daily was given orally for 3 consecutive days.

DISCUSSION

Ketosis is a common disease of adult cattle. It typically occurs in dairy cows in early lactation and is most consistently characterized by partial anorexia and depression. Rarely, it occurs in cattle in late gestation, at which time it resembles pregnancy toxemia of ewes. In addition to inappetence, signs of nervous dysfunction, including pica, abnormal licking, incoordination and abnormal gait, bellowing, and aggression, are occasionally seen (Hibbitt, 1980). In this case study inappetence and low production was the common findings

The pathogenesis of bovine ketosis is incompletely understood, but it requires the combination of intense adipose mobilization and a high glucose demand. Both of these conditions are present in early lactation, at which time negative energy balance leads to adipose mobilization, and milk synthesis creates a high glucose demand. Adipose tissue mobilization is accompanied by high blood serum concentrations of nonesterified fatty acids. During periods of intense gluconeogenesis, a large portion of serum nonesterified fatty acid is directed to ketone body synthesis in the liver. That means when large amounts of body fat are utilized as an energy source to support production, fat is sometimes mobilized faster than the liver can properly metabolize it. If this situation occurs, ketone production exceeds ketone body utilization by the cow, and ketosis results (Grummer, 1993). In this case study, ketone bodies was found in the urine sample.

During ketosis glucose level become lower than the normal level. Normally animal synthesis glucose from carbohydrate. At first carbohydrate convert to acetic acid, butyric acid and propionic acid. This propionic acid convert to oxal-acetate than glucose. During lactation loss of glucose and result hypoglycemia. In ketosis, lack of oxalo acetate. In absence of the oxalo acetate, acetic acid, butyric acid converted to aceto acetic acid that results ketosis. Normally in presence of oxalo acetate, acetic acid and butyrid acid provide energy via try carboxylic acid cycle. Hypoglycemia, ketonemia and ketonuria are the characteristics of the disease (Adler and DYE,1995). In this case study the serum glucose level was 40mg/dl which was lower than the normal range. Ketonuria also observed in our study. This all findings indicate that cow is suffering from ketosis.

Ketosis either is clinical or subclinical. It is defined according to presence or absence of clinical signs. Two major forms of clinical signs are described. One is wasting form and another is nervous form. The wasting form is the most common of two and is

manifested with gradual but moderate decrease in appetite and milk yield. (Esposito, 2014). In this study we found that the cow lost the appetite gradually and production also decreased day by day. So this indicates the cow was suffering from ketosis.

It is important to prevent ketosis from occurring, rather than treating cases as they appear. Prevention depends on adequate feeding and management practices. In times of feed deficiency because of drought or other reasons, the provision of supplementary feed with adequate amounts of carbohydrate is essential. The best feeds tend to be good quality hay, silage, or cereal grain. The body condition of the dairy cow is important at calving. Cows should be on a rising plane of nutrition up to calving with the aim to calve in good condition. After calving, the cow has the potential to reach maximum efficiency in milk production, but feed requirements for high production are often greater than the voluntary intake of pasture can provide. Therefore an energy supplement is required and there is evidence that this will improve production and reproductive performance, and decrease the risk of ketosis. The best supplements are good quality hay, silage, or cereal grains. Supplements should be fed at least until the peak of lactation is reached or longer depending on the quality and quantity of available pasture (Oetzel, 2004). The owner was advised to supply balance rations to all the lactating cows of his farms in order to prevent ketosis.

CONCLUSION

Ketosis is an important disease in dairy cattle farms which result in great mentionable financial damages to milk production due to its effects on health or production of dairy cattle. It is a metabolic disorder. During this time energy demands exceed energy intake and results negative energy balance. Ketotic cows have low blood glucose concentrations. Large amount fat utilize for energy source to support production. Sometime fat mobilize faster than the liver can metabolize it. Clinical signs of ketosis are reduction of milk production, loss of weight, reduction of appetite. As production decrease we need to give more emphasis on regular monitoring providing balance diet, regular screening of milk and urine for ketone bodies detection, regular evaluation of body condition score are the best methods of early detection of ketosis in animal. This can be easily applicable in dairy cattle farm. We need awareness to prevent this in the farm level.

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Biography

I am Md. Muhyminul Islam son of Abdus Samad Mridha and Khudeja Begum. I passed Secondary School Certificate examination in 2011 (G.P.A 5) followed by Higher Secondary Certificate examination in 2013(G.P.A 5). Now I am an intern veterinarian under the Faculty of Veterinary Medicine under the Faculty of Veterinary Medicine in Chattogram Veterinary and Animal Sciences University. In the future I like to work as a veterinary practitioner and do research on clinical animal diseases in Bangladesh.