DIAGNOSIS AND MANAGEMENT OF METABOLIC DISORDERS IN HIGH YIELDING DAIRY CATTLE

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Introduction

Recently with rapid demand of high producing cows and with challenging feeding system, the dairy cow have been found to be associated with metabolic disorders. It is a highly complex and a number of associated, interrelated conditions. The incidence of metabolic disorder is the highest at time of calving and extending until the peak lactation is reached. Identification of etiology is valuable aid to diagnose the metabolic disorders in most economic way. So detailed investigation are taken up-to study the diagnosis of those disorders and to evolve a specific and suitable management strategies to prevent it to reduce economic losses of a dairy herd.

Ketosis

Ketosis is an increase of ketone bodies (acetone, acetoacetic acid, and Beta-hydroxybutyric acid) in blood until they eventually begin to spill over into urine and milk. In dairy cows, ketosis is a lactation disorder usually associated with intense milk production and negative energy balance. Diagnosis can be made by sign which appear abruptly and include loss of appetite, particularly for concentrate feeds, decrease in milk production, and rapid loss of body condition. Some cows become excitable although majority is apathetic. Body temperature remains normal. The milk gives a positive reaction to the Rothera test (modified). The normal level (mg/ 100ml) of acetoacetic acid, Beta-hydroxybutyric acid and free fatty acid in high yielding dairy cows are 0.1, 8, 9 but during ketosis levels are reached to 7, 30, 28, respectively.

It is very common that during dry season animals are generally poor fed leading to absolute lack of carbohydrate. The high lactation demand seems conducive to excess fat mobilization, which contributes to development of ketosis. In some cases ACTH or cortisone deficiency and deficiency of oxaloacetic acid in liver are primary cause of ketosis. Secondary ketosis include mastitis, metritis, displaced abomasum, indigestion, retained placenta, nephritis and extended milk fever. Differential diagnosis must be carried out in following cases viz : digestive form of ketosis may be confused with some diseases like indigestion, traumatic reticulitis, abomasal displacement, pyelonephritis, vagus indigestion, metritis, cystic ovaries, pleurisy, pneumonia, peritonitis, diabetes mellitus and nervous form of ketosis may be confused with rabies, lead and nitrate poisoning etc.

MANAGEMENT :

Following Management strategies can be followed to to reduce the Ketosis.

- Avoid poor quality silage
- Supply propionate, vitamins and minerals, protected fat, ionosphere, niacin (3-6 g/d).
- Provide Somatotrophin injection during later part of lactation and chloral hydrate @ 7 gm twice daily for 3-5day (Baird 1982).

Schultz, (1984) reported to practice the following.

- Increase concentrates intake in late dry period.
- Provide 1/3 of DM as high quality rough.
- Avoid abrupt dietary change and stress.
- Provide exercise and comfort.
- Monitor milk ketone body at weekly interval.
- Provide following feed additives (Littledike et al, 1981).

ADDITIVES

Level (gm)

1 – 3 Butandial or Propylene glycol

225 gm twice daily

Ammonium Lactate

200 gm daily for 5 days.

Na – Propionate110 gm daily for 6 week at calving.

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● Following treatment may be given.

DRUG

Dose	
Dextrose (40 – 50%)	500 – 800 ml
Glycerence	100 mg twice daily for 2-3 days
Betamethasone Injection	30 mg – I/M

Milk Fever

Milk fever is an afebrile hypocalcemic disorder of cattle usually associated with parturition and initiation of lactation. Diagnosis is based on conditions of paresis and depression in animal that have calved recently. Symtoms which are helpful for diagnosis are excitement, tetany with hypersensitiveness, muscle tremor of head and limbs, protrusion of tongue, grinding of teeth, stiffness in hind legs and stuck out stiffly, sternal recumbence, drowsy appearance and head turned into the flank, rectal temperature subnormal, pupils are dilated, loss of anal reflex and relaxation of anus, marked decline in intensity of heart sound, weak pulse, ruminal stasis, constipation. Death occurs due to cessation of respiration or convulsion. The diagnosis is confirmed by a favorable response to treatment with parental injections of calcium solutions and by biochemical examination of blood. The level (mg/dl) of calcium in normal is varies from 9 to 11, whereas in milk fever cases level goes down to 2 to 5. The Ca, P and Mg concentration in blood of parturient cow is depicted in Fig 1. Differential diagnosis can made by identification of similar type of diseases which cause recumbence like calving injury, rupture of joint/ ligaments of joint, septic metritis, vaginitis, rupture of uterus, toxaemic condition and drowner cow syndrome etc (Chakrabarti 1988).

MANAGEMENT :

Following Management strategies can be followed to reduce it.

● ***Nursing i.e calf should be removed after 48 hrs, gradual return to full milking, moves the animal from side to side for 3-4 times, shelter over cows.***

Inflation of the udder is also an effective treatment.

● Following prepartum dietary management (Gayner, 1989) can be practiced.

-Alfalfa haylage + Choloride (Mg, NH_4, Ca) salt.

-P, Ca (100-125 g/d), MgO (60 g/d), NH_4Cl (23 –25 g/d), Ca + Vit – D_3 (8 mg – I/M).

-At calving – Ca salt in gel @ 100 g/d.

-Avoid over fattening and stress.

● One effective method is to feed a low Ca diet at least 5 days before parturition (Green et al, 1981). This procedure causes a slight hypocalcemia and increased secretion of parathyroid hormone. Under continued stimulation of these two hormones, bone tissue is prepared to meet much of Ca demands of lactation so that hypocalcemia do not develop.

● Following Treatment may be given.

Drug Route	Dose
Ca – Borogluconate or Ca-Mg – Borogluconate (20-30%) 200 – 300 ml	I/V or S/C
Na- Acid phosphate1 oz	I/V
& Glucose 4 oz	I/V
Avil or Phenergan10 – 15 ml	I/V

GRASS TETANY

Grass tetany is caused by inadequate magnesium in critical extracellular fluids and hypocalcemia is generally associated with it. Diagnosis can be made by symptoms like affected animals become restless, stop grazing, twitching of muscles and ear, frenzied galloping, staggering gait serum Mg levels fall below 1 mg/100 ml compared with normal level of 1.7 to 3.0 mg/100 ml, frothing at mouth, pricking of ears and retraction of eye lids, temperature rises after severe muscle exertion, pulse and respiratory rates are also high. Death usually occurs within ½ to 1 hr and mortality rate is high. Incoordination, hyperaesthesia and tetany may be confused with acute lead and nitrate poisoning, rabies, nervous form of ketosis and ergotism etc. Differential diagnosis must be carried out in these cases. Tetanogenic pasture, sandy latarite soil, cereal grazing and also starvation, hypothyroidism reduces the Mg level in animal (Robinson et al 1989). Major factors associated with reduced availability of Mg which is ingested are enumerated below :

(1) Heavy top dressing of pasture with Ammonia fertilizer reduces up takes of Mg by plants and subsequently reduced availability of ingested Mg due to NH_3 in rumen.

(2) The presence of chelating agent keto-butyric acid in plants and rumen.

(3) High K content of pastures may reduce absorption of Mg. The grass with high ratio of K to Ca and Mg are more likely to cause grass tetany.

(4) Potassium is known for its competition with Na for absorption in plants interferes with Mg absorption.

MANAGEMENT :

Following management strategies can be followed to reduce it.

1. Feeding of Mg salt during danger period, 60-120g/day.
2. Several other salts, e.g., MgO, Mg acetate, Mg phosphate with molasses which can be fed as free access mixture.
3. Heavy Mg bullets to be put in reticulum where it releases 1g Mg/day.
4. Top dressing of pasture with Mg rich fertilizer raises Mg in pasture and reduces susceptibility. Calcined Magnosite – 1125 kg/ha or Mg limestone 5600 kg/ha may be used.
5. Spraying of 20% solution of $MgSO_4$ can raise level of Mg much more quickly at fortnightly intervals.
6. Increase legume, reduce K application and add Mg fertilizer in field.
7. Avoid calving during months when hypomagnesemia is likely to occur.
8. Treatment are given below.

Drug	Dose (ml)	Route
Mifex	100 – 150	I/V
$MgSO_4$ (10-20%)	200 – 300	S/C
Mg- Gluconate (15%)	200 – 300	S/C
Mg – Adepate (12%) + Ca – Gluconate (5%)	250 + 250 each	S/C

HYPOPHOSPHATAEMIA

It occurs after 2-4 weeks of caving in high producing dairy cow. Diagnosis can be done by symptoms viz : Haemoglobinuria, haemolytic anaemia, subnormal temperature, jaundice and dehydration. Estimation of phosphorus level in blood which indicate that normal level (mg/dl) is 4 -7, were as in hypophosphataemia case level goes down to 0.5-3. History of advanced pregnancy or recent calving will also helpful. If low level of inorganic 'P' in blood is replaced by supplementation of P animal will respond and thus help in diagnosis of the disease. Differential Diagnosis have to be done from similar type diseases viz: Leptospirosis, Bacillary haemoglobinuria, Babesiasis, Anaplasmosis, drug induced such as phenothiazine, Enzootic haematuria, Blood-transfusion reaction, Pyelonephritis, Myoglobinuria, Chronic copper poisoning.

MANAGEMENT :

- P Supplementation

Bone Meal or Decaphosphate 120 gm (twice daily)
Na – Acid Phosphate 80 gm in 400 ml dist water – I/V
Inj. Tonophosphan 10-15 ml/day – I/M for 4 to 5 day

- **Correction of anaemia**

(1) Blood transfusion – 5 lit for 450 kg bw. (2) Fluid therapy, (3) Haematonic mix containing Ferric sulphate (5gm) + $CuSO_4$ (0.2 gm) + $CoSO_4$ (0.2 gm), daily for 10 days, (4) Inferon Injection and (5) Liver extract can be fed orally.

Downer Cow Complex

Downer cow diseases are complications resulting from parturient paresis with demonstrable muscle, tendon, or nerve injuries. It can be diagnosed by observing the following aspects viz: traumatic injuries in medial thigh, obturator muscle, hip joint and or prolonged recumbence for more than 4 to 6 hr which lead to downer cow complex. Low milk production and prolonged calving intervals also promote it. Feeding excessive quantities of concentrates after peak lactation or during dry period combined with free choice feeding of corn silage or high quality hay predisposes a herd to development of this disorders. Likewise, either underfeeding of protein or an early decrease in milk production, combined with free-choice feeding of corn silage, contributes to excessive intake of energy and over conditioning of cows lead to this disorder.

MANAGEMENT :

Early detection and treatment of milk fever at first stage, correct P and K deficiency, administer dextrose (5%) with electrolyte @ 500 to 1000 ml (I/V), avoid slippery ground surface, recumbent cow should be well bedded on soft ground/straws, rolled from one side to another at regular intervals, gradually assist to stand are most helpful to manage the downer cow syndrome in economic way.

ACIDOSIS

It can be diagnosed by history of excessive intake of readily fermentable carbohydrates, which causes selective proliferation of lactic acid producing microorganisms in the rumen with results that concentration of individual acids, particularly lactic acid increases and acidosis formed. Any foodstuff with high content of readily

available carbohydrate can cause acidosis. A lower P^H of rumen contents is also possible when proportion of crude fibre is reduced. Poor mixing of grain and faulty feeding may lead to this disorder.

MANAGEMENT :

1.Recently it has been reported that inoculation of rumen contents by Megasphera elsdenii almost completely reduces the risk of acidosis in animals fed high concentrate diet (Robinson et al., 1992).

2.Gradual adaptation to easily digestible food over a period of 20 days by slowly increasing concentrate feed and feeding in three equal installments per day.

3.Maintaining a minimum level of crude fiber in ration (18% for milking cows) and supplementation with $NaHCO_3$ or other buffers (Erdman et al., 1980).

4.The presence of yeast cultures resulted in a significantly lower level of lactic acid.

5.Provide Thiamine hydrocholoride (2 to 4 gm – I/V), liver extract along with Vit B (3-5 gm /day).

6.Ruminal message along with rumentorics is also helpful.

LOW MILK FAT SYNDROME

The secretion of normal volume of milk but its fat content reduced, often to less than 50% of normal is a significant cause of wastage in high producing cows. It occurs in cows fed low fibre diets. Diagnosis can be made by essential features of most widely tested diets that depress milk fat are – (1) A high ratio of readily digestible carbohydrate to fibrous constituents. (2) Dietary supplements of certain oil and unsaturated fatty acids to another normal diet. The degree to which lactating cows respond to these diets depend on factors such as physical preparation of diet, level of feeding or frequency of feeding, stage of lactation, level of milk production and fatness in animal. (3) Increased gluconeogenic activity consequent to higher production of propionate in rumen reduces insulin level and results in reduced mobilisation of lipids from adipose tissue which in turn has been shown to be responsible for low fat milk production, by affecting the activity of glycerol phosphate dehydrogenase.

MANAGEMENT :

Following management strategies can be adapted to avoid it.

(1) Feeding of 4% of 1, 3 butendiol largely corrects the decline in milk fat by providing Acetyl CoA.

(2) Restoration of proper diet containing sufficient roughage can help normal level of fat in milk.

FAT COW SYNDROME

Fatty infiltration of liver occurs in cows most commonly within first two weeks after parturition and is associated with parturient disease. Diagnosis is very difficult but history, breed, obesity, grain feeding, lab exam should be to taken up. This disorder may be confused with abmosal displacement, ketosis, parturient paresis and downer cow syndrome. It may also be confused with vagus indigestion, and chronic peritonitis if the disorder is occurred before calving. Differential diagnosis carried out in these cases. The mobilization of excessive quantities of fat from adipose tissue to liver because of sudden demand of energy during post-partum period in lactating dairy cows is said to be the cause.

Management :

Following Management strategies can be practiced to avoid this disorder.

(1) Maintain appetite and prevent pregnant cow from becoming fatty during last trimester of pregnancy.

(2) Control reproductive and post reproductive disorders.

(3) Sorting the animal into groups on basis of body condition which is helpful for following proper feeding practices.

(4) The oral administration of propylene glycol (100 mg/day) will promote glucose metabolism.

(5) The use of insulin 200 to 300 i.u. S/C twice daily will also help.-

(6) Provide good quality hay and ample supply of water.

(7) Water and electrolytes should be administered intra-ruminally.

(8) Choline chloride at 50 gm/day and ruminal juice (5-10 lt), propylene glycol can also be given.

(9) Dextrose (50%) @ 500 ml through I/V can be given.

Conclusion

In modern dairy industry metabolic disorders are getting much economic importance. The occurrence of these disorder varies from season to season, year to year, region to region. During the time of parturition, fat cows appear to be more susceptible. Only through better management it will be possible to prevent the occurrence of these metabolic disorders. ❑