Production diseases - an overview

R.Vijayan

Dr.R.Vijayan
Associate Professor,
Department of Animal Husbandry
College of Agriculture,
Vellayani,
Thiruvanathapuram.

The term production diseases comprises not only diseases previously known as Metabolic diseases but also some other conditions which adversely affect production either directly or as predisposing factor. But all these conditions are etiologically related to the diseases of metabolism. Even though the general term production diseases is agreeable, many practitioners proposes to continue the usage of metabolic diseases because of its common usage.

High prevalence of production disease among cross bred cattle of Kerala has been recorded in the period commencing at calving and extending until the peak of lactation. Pregnant ruminants also often become victims of production diseases. Milk fever and Ketosis dominate among the production diseases prevailing in cross bred cattle of Kerala.

After parturition there is a sudden onset of a profuse milk yield in which the nutrient reserves will be seriously depleted. The essential metabolite which is reduced below the critical level will manifest the clinical syndrome. Predicting the occurrence of production diseases in advance as preventive measure is the current trend. The metabolic profile text is based on this concept and mixed feelings expressed about the practical usefulness of the system.

All these metabolic disorders except udder edema were directly interrelated. In the same manner, variations in the type of diseases occur. This creates problems in the medical management of the metabolic diseases.

A depression of the levels of ionized calcium in tissue fluids is the basic defect in milk fever. It is not always associated with an absolute deficiency but is believed to reflect a temporarily impaired ability to mobilize reserves with in the animals.

Some degree of hypocalcemia occurs in nearly all cows at the onset of lactation. Hypocalcemia stimulates the calcium homeostatic mechanism to improve the efficiency of intestinal calcium absorption and increase bone resorption. If these adaptations to increase calcium demand are prolonged, clinical hypocalcemia may be developed.

Cows with parturient paresis are usually hypophosphatemic (as low 2.1 mg/d) hypermag nesemic (serum magnesium of 2.2 t0 2.7 mg /dl) and hyper glycemic (95 to 130 mg/dl) Unlike parturient paresis, non parturient hypocalcemia is frequently associated with hypomagnesemia and hyper phosphatemia.

In the past, low calcium diets fed in the dry period were the primary method of milk fever prevention. It is now known that this method is not as effective as initially believed. It is also difficult to get a low amount of calcium in the diet (less than 20 g of calcium per day)

Using the dietary cation-anion balance (DACB concept) has revolutionized the prevention of parturient paresis. The concept of DACB in dairy rations was based on the observation that diets high in cations, especially sodium and potassium tend to induce milk fever compared with those high in anions which prevented the incidence of milk fever. The key to this

method in providing an excess of anions over cations in the prepartum diet by adjusting the diet, adding anionic salts (sulphate and chloride) or a combination of both. Anionic salts that can be added are magnesium sulphate, calcium sulphate, ammonium sulphate, ammonium chloride and magnesium chloride.

Administration of Vit D3 and its metabolites, oral calcium gels or drenches administrated at the time of calving and intravenous synthetic bovine PTH (if administration is begun atleast 60 hours before parturition) are effective in preventing parturient paresis.

The input must be equal or exceed the out put to prevent a negative energy balance. When milk production exceeds the capacity of the animal to ingest sufficient feed to meet requirements for energy will lead to negative energy balance. This occurs with crossbred cattle of Kerala because genetically the animal have high milk yield capacity. To offset the negative energy balance, the individual cow must mobilize body fat and protein sources for gluconeogenesis.

Normal high producing cows will have some level of ketosis during early lactation until their energy intake balances milk production. During this period cow lose 30 to 100 kg body weight. The difficulty is in identifying and preventing the factor that move a cow from the normal level of ketone body formation into subclinical and clinical categories

Prevention and control of ketosis can be taken up in three steps

1. The body fat that is lost in early lactation must be stored in the previous late lactation by feeding. It is essential that cow should not become too fatty before calving.

2. Following calving, incremental increase are made in feeding cattle.

3. Subclinical and clinical ketosis should be detected and treated as early as possible. This may be accomplished by encouraging clients to use ketone tests routinely on milk or urine during first 50 to 60 days following parturation.

The essential finding in the downer cow syndrome were that all cows had an initial clinical episode suggestive of milk fever but showed an unsatisfactory clinical response even after successive calcium therapy.

A high incidence of downer cow syndrome had been observed among the cross bred cattle of Kerala. Medical treatments to this syndrome has not been noticably successful Many treatments are attempted empirically in the downers without consistent success.

Hypomagnesemia is a magnesium ion deficiency of the blood and cerebrospinal fluid. It is highly fatal and affecting only ruminant species. Hypomagnesemia is only usually accompanied by hypocalcemia. Treatment is often not successful if the cow is already comatose.

Since exogenously administered Mg equilibrates slowly across the blood brain barrier as many as 20 percent of the treated cows die during convulsion despite prior therapy. Relapses are common within 3 to 6 hours of treatment.

Fatty liver syndrome is a common problem and is due to a generalized mobilization of fat from body depots to the liver. There is a rapid loss of body weight. This produce a syndrome in which the affected cattle are susceptible to metabolic, infectious and reproductive problems. This conditions has been called pregnancy toxemia of cattle.

The affected cow do not respond to treatment and become totally anorexic. She may become recumbent and develop a severe form of ketosis, which does not respond to the usual form of therapy and die in 7 to 10 days. In general cows which are totally anorexic for three days or more will die, those which continue to eat even a small amont will recover with supportive therapy and nutrition.

The secretion of a normal volume of milk but with its milk fat reduced often to less than 50 per cent normally is described as low milk fat syndrome (LMF). LMF is influenced by several factors including nutrition, temperature humidity , genetics and cows state of lactation. Feeding high grain low roughage rations, finely chopped forages, reduced size of hay low fibre diet are described as dietary causes of LMF.

Occurrence of metabolic diseases varies from season to season and from year to year. Disturbance of more than one of the metabolites mentioned may occur simultaneously in one animal and give rise to complex syndrome which are not described here. More common disease entities alone are described but other imbalances are also important.