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Fatty Liver Syndrome in Dairy Cattle

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Abstract

Fatty liver is the accumulation excess of non essential fatty acids (NEFA) in liver from adipose tissue in order to compensate the negative energy balance after parturition in dairy cows. It is also associated with other metabolic diseases like milk fever, ketosis and infectious diseases like mastitis and salmonellosis. Failure to reduce energy intake after peak lactation leads to over conditioning of cows in the dry period and it ends up with fatty liver. Treatment for this condition includes providing a balanced diet, symptomatic treatment depends on condition and administration of choline chloride, dextrose and vitamins. Avoid rapid diet changes, unpalatable feedstuffs and environmental stress because they may leads to reduction in feed intake and trigger catecholamine-mediated increases in fat mobilisation. By providing good feeding management like separation of cows in dry period and reducing the energy and concentrate feed intake we can prevent the over conditioning of cow and fatty liver disease in dairy cows.

Currently India is the number one country in the milk production 53.77 Million Tonnes (2017-18) with 29.9 crores of bovine population (2012 census). As human population increases, the consumption of milk also increases. So the milk production volume is increased very fast because of animal reproduction, nutrition, breeding, prevention of disease and advances in management. In the meantime the rate of occurrence of metabolic diseases also increased in dairy cows. Among them fatty liver disease

is the one which mainly affects the health and production performance of dairy cows. Fatty liver is a nutritional disease of ruminants due to the accumulation of fat or triglycerides in the liver (Kato, 2002). It is also called as fat cow syndrome and pregnancy toxemia. The syndrome not only affects milk production, but the animals often suffer from various metabolic diseases such as milk fever, ketosis, digestive disorders such as displaced abomasum, infectious diseases such as mastitis and salmonellosis and reproduc-

tive conditions such as retained fetal membranes and metritis (Morrow, 1976). This combination of disease conditions in the obese periparturient cow has been termed the fat cow syndrome. The metabolic diseases such as ketosis, milk fever and downer cow syndrome are the most common expensive disease entities in lactating dairy animals. This disease condition cause severe economic losses in terms of heavy reduction in milk yield and impaired reproductive performance (Senthilkumar *et al.*, 2013).

According to the most recent National Animal Health Monitoring System for dairy cattle (National Animal Health Monitoring System, 2008), leading causes of morbidity in dairy cattle are clinical mastitis, lameness, infertility, retained placenta, milk fever, reproductive problems, and displaced abomasum. Of cows removed from herds, about 53% leave for one or more of the above reasons (Beitz, 2014). Sources of economic loss in fatty liver diseases includes mortality, decreased milk production, impaired reproductive ability, veterinary care for periparturient problems, infectious diseases, reproductive failures and feed wasted by overfeeding heifers and dry cows. Fatty liver lead to scarring and hardening of the liver and death of the cow as a consequence. Without treatment mortality can be as high as 25% (Ametaj, 2005).

The Cause

Over conditioning of dairy cows in the pre-partum period is the main

cause of fatty liver disease. This is due to the excessive intake of an unbalanced diet mainly feeding excessive quantities of concentrate combined with free choice feeding of corn silage and/or hay crop forages during both late lactation and dry period. Lead feeding at parturition and failure to restrict concentrate intake after peak lactation predisposes cows to obese (Morrow, 1976). Fatty liver occurs as a result of hepatic uptake of nonesterified fatty acids (NEFA) that are released in excess by adipose tissues attributable to negative energy balance. Thus the liver becomes fat when the cow is losing condition, the more loss in condition the more fat in the liver. Dry matter intake reductions along with high energy demand for milk production are the main risk factors for fatty liver (Gonzalez and Rosendo, 2013).

Occurrence

Fatty liver disease occurs sporadically, depending on feed management practices. Usually the few days following parturition is the critical time for the occurrence of this condition. It occurs most frequently in loose housing system where cattle in all lactation stages are fed and managed in one group (Haass and Eness, 1984). The time requirement for the development of this disease depends on the initial condition of the cows and amount of high energy feed available for consumption in free choice. Most metabolic diseases in dairy cows occur during the peripartum period (2 to 4

weeks after parturition) are suggested to be derived from fatty liver (Katoh, 2002). Fatty liver can develop within 24 hours of an animal going off feed. This is typically around the calving time. There may also be an inherited tendency to develop fatty liver.

Pathogenesis

The four stages of the lactation cycle of cows are early lactating, mid lactating, late lactating and non-lactating. The milk yield is highest during mid-lactation and cows during this stage are fed high-energy diets. Feeding of such diets tends to continue even during late lactation, mainly in high yielding cows. This leads to shortening and over feeding the non-lactating stage and cows become obese before parturition. The feed intake is reduced near parturition meanwhile the demand for energy progressively increased by initiation of lactation after parturition. To overcome this negative energy balance after parturition fat from the adipose tissue gets breakdown and nonesterified fatty acids (NEFA) mobilized from adipose tissues. It is transported by binding with serum albumin and is mainly taken up by the liver (Bell, 1980). Then NEFA in liver is converted to triglyceride and is secreted as very low-density lipoprotein (VLDL) or, alternatively, is oxidized in mitochondria and peroxisomes. In cattle the major site for fatty acid synthesis is adipose tissues, not the liver (Bell, 1980). Ruminants due to the lack of adequate hepatic lipoprotein lipase and hepatic

lipase, hydrolysis to remove triglycerides approach is limited obviously, VLDL secretion is the main way of liver removal of triglycerides. The ability of ruminants to secrete hepatic triglyceride as VLDL is extremely low compared with non-ruminant animals (Pullen *et al.*, 1990). When the amount of incorporated NEFA exceeds the amount secreted as triglyceride by and oxidized in the liver, triglyceride accumulates in the liver and fatty liver develops (Katoh, 2002) (Gerloff *et al.*, 1986). Accumulated triglyceride impairs the metabolism of lipoproteins, most of which are produced by the liver. Lipoproteins have a role in the transport of lipids between the liver and extra hepatic tissues. The impairment in turn influences other metabolic pathways in extra hepatic tissues. Impaired liver results in low serum albumin and reduced liver metabolism of sex steroids which is associated with reduced fertility in cows. Fatty infiltration in other organs increases the cow's susceptibility to other infectious and metabolic diseases.

Clinical signs

The syndrome has no unique clinical signs. The main symptom exhibited in the fat cow is a refusal to eat. Excess fat in and around the internal organs and on the cow can reduce feed intake after calving possibly by restricting gut capacity and by the release of larger than normal amounts of free fatty acids into the blood, thereby depressing appetite (Harris,

1992). The other clinical signs include depression, ketonuria, marked decrease in production, progressive debilitation, excessive loss of body condition, weakness, nervous signs, reduced fertility and an elevation in temperature due to infectious disease (Morrow, 1976) (MSD Manual Veterinary Manual, 2019). There is great chance of occurrence of milk fever, ketosis, clinical mastitis and retained fetal membranes.

Diagnosis

The diagnosis of the fat cow syndrome is based on a history of excessive energy intake. Obese cows and the presence of one or more periparturient conditions such as milk fever, ketosis, displaced abomasum, retained fetal membranes, and/or mastitis are diagnostic (Morrow, 1976). Liver biopsy is a minimally invasive procedure that is the only direct and most reliable method to determine severity of fatty liver in dairy (Kirovski and Sladojevic, 2017). However, these assays are not routinely conducted in commercial laboratories. If hepatic triglyceride contents of the peripartum cow is more than 30 mg/g of liver it is defined as fatty liver (Kato and Kimura, 1989). Moreover, determination of the apolipoprotein concentrations and enzyme activity during the peripartum period is useful for early diagnosis of this disease. Blood tests will show reduced glucose concentration, elevated levels of nonesterified fatty acid concentrations (NEFA) and increased ketones. If concentrations of

NEFA > 0.7 mM in first week of postpartum period the animal must have problems with energy balance (Kirovski and Sladojevic, 2017). The prognosis is guarded to poor.

Treatment

Treatment of the condition consists of feeding a balanced diet, symptomatic treatment, and good supportive care. The general treatment is usually symptomatic for the periparturient conditions like cows with milk fever are treated with calcium preparations and ketotic cows can be administered with dextrose and nicotinic acid. The ultimate aim for this therapy is to inhibit fat mobilization which will decrease the quantity of fatty acids presented to the liver. Choline chloride (50%) can be administered orally at 50 to 100 g once daily (Morrow, 1976). Choline is essential for phospholipid formation which is required for lipoprotein production and fat transport (lipotropic agent). Since methionine is a precursor of choline, it also should be beneficial in treating this condition (Morrow, 1976) (Best and Lucas, 1962). Other treatments includes administration of intravenous glucose (10% or 50% dextrose) and electrolyte solutions, insulin which is antilipolytic, anabolic steroids and propylene glycol by drench to stimulate rumen motility (Haass and Eness, 1984). Vitamin K is important in the management of animals with liver disease because normal liver stores vitamin K, which may become significantly depleted in liver disease. Oral cobalt sul-

fate solution and/or B complex vitamins are commonly used to stimulate the appetite. Broad spectrum antibiotics should be administered in therapeutic daily doses to control infection which is frequently in the form of mastitis and metritis. Rumen transplants and feeds high in readily digestible nutrients should be fed in the amount of 11 to 21 liters twice daily until the appetite is restored (Morrow, 1976).

Prevention and control

Management of cows in dry milk stage is very important to prevent the fatty liver disease. The ideal body condition score at calving stage is 2.5 to 3. Cows should be maintained at this score when drying off, and weight should be maintained throughout the dry period. Shortening of dry period 45 to 60 days or maintaining the calving interval 12 to 13 month is considered to be optimum for best efficiency and milk production (Morrow, 1976). It helps to prevent cows from becoming too fat. Exercise during the dry period also reducing the incidence of fatty liver (Blake *et al.*, 1975). Cows in loose housing should be grouped by production to feed them according to requirements for milk production (Haass and Eness, 1984). Dry cows should be separated after peak lactation period and the concentrate intake and all high energy feed intake should be limited during the dry period. The energy intake can be limited by feeding coarse grass hay or pasture which also will help prevent milk fever and displaced abomasum. It is important that the

ration for dry cow should be balanced for energy, protein, vitamins, and minerals to utilize nutrients to maximum efficiency and to prevent disease. Avoid rapid diet changes, unpalatable feedstuffs and environmental stress because they may leads to reduction in feed intake and trigger catecholamine-mediated increases in fat mobilisation. High grain feeding prepartum depresses total dry matter intake at parturition (Coppock *et al.*, 1972). So challenge feeding rather than lead feeding of grain should be practiced prior to calving. This procedure consists of feeding approximately 2.3 to 4.5 kg of grain for 2 week prior to calving to permit the rumen protozoa and bacteria to adapt to the change in diet (Morrow, 1976). Propylene glycol, 300–600 ml/day, given as an oral drench during the final week prepartum will reduce plasma NEFAs and the severity of fatty liver effectively at calving (MSD Manual Veterinary Manual, 2019). Administration of bile acids in dry period also showed prevention and control of this disease by the following mechanism. Free cholic acid combined with farnesoid X receptor (FXR) which suppresses the expansion of SREBP-1c, a critical transcription factor that regulates hepatic TG synthesis by inducing key enzymes involved in lipogenesis, such as fatty acid synthase (FAS). By inhibiting SREBP-1c expression bile acids control the proportion of fatty acid synthesis and by inducing the expression of PPAR α it increase the oxidation of fatty acid, thus

reducing hepatic steatosis. As a cofactor bile acid enhance the capacity of VLDL removal, excess triglycerides transport out of the liver and improves the intestinal bile acid circulation, liver function, improvement of par-enteral nutrition can cause liver disease, which reduce the extent of fat liver (Watanabe *et al.*, 2004).

Finally to reduce the incidence of fatty liver, first priority should be given to feeding a balanced ration to meet nutrient requirements and follow the recommended management to prevent metabolic, digestive, infectious, and reproductive conditions which contribute to the fat cow syndrome in the obese periparturient cow.

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