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An overview of fatty liver development and prevention during transition period in high milk yielding dairy animals

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Introduction

Fatty liver is a metabolic disorder seen in dairy cattle with high milk yield often developing during transition period due to the increased unmet energy demands of the animal. In this disorder, due to negative energy balance, animal body fat is broken down to produce non esterified fatty acids (NEFA) and then re-esterified in the ruminant liver to form fat or triacylglycerol (TAG). Due to a gradual efflux of fat or TAG from the liver, TAGs are accumulated in the liver and the functions of liver are impaired. The condition develops secondary to other comorbidities associated with pregnant animals or metabolic disorders and causes loss of productivity and poor metabolic condition of animals due to loss of liver function (Figure 1). It is also known as Fat cow syndrome or Hepatic lipidosis.

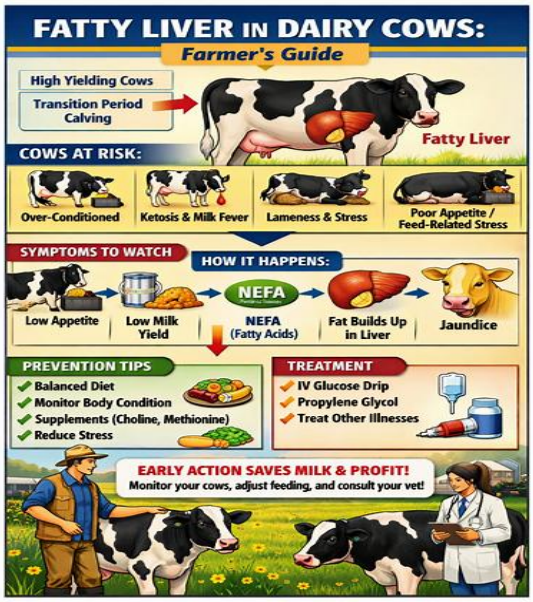

Figure 1: Insights into Fatty Liver development and prevention in high milk yielding Dairy Animals (Open AI, 2026)

Predisposed animals

Breeds selectively bred for high milk production like Holstein Friesian are highly predisposed to fatty liver condition during dry period (Shi et al., 2020). Overconditioned animals with body condition score (BCS) >3.5 during the transition period are the most vulnerable to the development of fatty liver (Rukkwamsuk et al., 2000). Animals suffering from metabolic conditions like ketosis, hypothyroidism are predisposed to it. Conditions which disable the animal from feeding like lameness due to fractures or recumbency due to hypocalcemia (milk fever) also act as precipitating factors for the development of fatty liver in cows.

Biochemical basis of development of fatty liver

Fatty liver develops in consequence to the steep gap between the metabolic energy requirements of the animal and the energy content of the diet which is termed as negative energy balance. The negative energy balance develops consequent to the sudden high demands of milk production confronted by the animal immediately after calving.

During the negative energy balance state, the animal body starts mobilizing the animal adipose tissue as fat can serve as a concentrated source of energy to meet the energy deficit (Klein et al., 2019). The enzyme lipase hydrolyzes the fat in the adipose tissue releasing NEFA. These EFA are taken up the liver cells readily as free fatty acids can be converted to ketone bodies or oxidized to produce energy via beta oxidation in the animal body.

However, when the amount of NEFA produced by adipose tissue fat breakdown surpasses the ability of the liver to convert it into ketone bodies or oxidise it to produce energy, excess NEFA enter another metabolic fate which involves their conversion back into fats or TAG within the liver cells. As such, the conversion of excess NEFA into fats inside the animal liver leads to accumulation of increased fat in the liver parenchyma (Figure 2). Although, liver does have an exit route for the excess fat by releasing it back to circulation by coating it with protein particles to form very low density lipoprotein (VLDL), this fate is compromised in ruminant liver as it is a slow process. As, the ruminant liver is not functionally able to release this excess fat in the form of very low density lipoprotein (VLDL), the trapped fat lodges inside the hepatic cells and compromises the metabolic functioning of the hepatocytes, culminating in fatty liver (Sejerson et al., 2012). Depending upon the extent of fat accumulation in the liver, the condition may be considered as mild (1-5 %), moderate (5-10%) and severe (> 10 %) (Bobe et al., 2004).

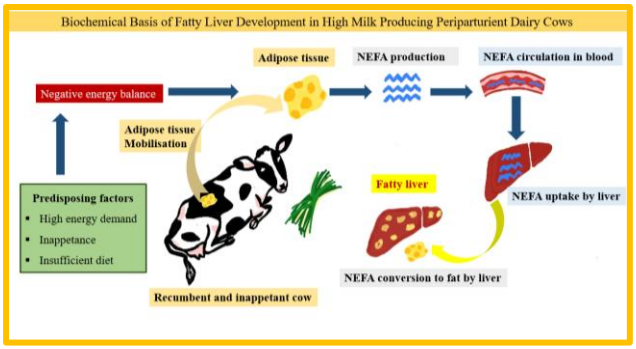

Figure 2: Schematic representation of biochemical basis of fatty liver development in high milk producing periparturient dairy cows. Under negative energy balance predisposed by high energy demand conditions, inappetence and insufficient diet, the adipose tissue is broken down to release non esterified fatty acids (NEFA). These fatty acids circulate in blood and are taken up by liver which converts them into fats responsible for formation of fatty liver.

Role of hormones in the development of fatty liver

The breakdown of adipose tissue is central to the development of fatty liver. A complex interplay of hormones underlies the development of fatty liver. The hormonal landscape of the peri-parturient animal is vastly different from a non-pregnant animal. During late gestation, because of production of placental lactogen, the insulin sensitivity of tissues is reduced, thereby creating a state of insulin resistance (Grummer, 2008). Insulin is required by the tissues to efficiently utilize glucose. But, the insulin resistance developed during the late gestation renders the tissues incapable of utilising glucose for meeting their

energy demands. This creates enormous onus on the adipose tissue to meet the raised energy demands. Additionally, during the last few weeks of pregnancy, cortisol levels are increased which exacerbate insulin resistance and increase lipolysis. As such, insulin resistance and increased cortisol together create a metabolic state which predisposes the animal to the increased lipolysis. This susceptible state of the animal coupled with insufficient diet or poor appetite leads to the development of this condition.

Symptoms and associated co-morbidities

Overconditioned animals are the most susceptible to the development of fatty liver and can develop this condition within 24 hours of going off feed. Cows with a mild form of the disease are hard to detect as they do not exhibit any specific clinical symptom except for reduced appetite and low milk yield. Due to disruption in hormonal signalling, the cows are less likely to show signs of estrus and have reduced fertility rates. Dairy cows also become susceptible to environmental toxins as the detoxification role of liver is impaired. In pregnant animals, associated conditions like metritis, mastitis, retained fetal membranes are often associated with cows developing fatty liver. (Melendez et al., 2022). Depressed appetite causes a reduction in rumination with accompanied abomasal displacement and ketosis in the animal (Duffield, 2000). Recumbency is also seen to be associated with the disease as recumbent downer cows often develop fatty liver due to prolonged periods of inappetence. In late stages of the disease, jaundice could also be evident due to the increased damage to the metabolic functioning of the liver. In severe cases of fatty liver, mortality is also observed.

Diagnosis

Fatty liver can be diagnosed on the basis of animal history, clinical findings, biochemical testing and ultrasonographic examination. History of the anorectic overconditioned animal with recent calving is suggestive of the condition. Rapid weight loss with decreased milk production after calving is noticed during this condition. Tests for presence of negative energy balance (estimation of NEFA, Ketone bodies and blood glucose) can be helpful in assessing the condition. Low blood glucose with high amounts of circulating NEFA's and ketone bodies (Beta hydroxy butyrate) are used as indicators for lipolysis. Hormonal profiling of thyroid hormones, insulin and cortisol can be done to evaluate the metabolic activity of the animal. Low levels of thyroid hormones with high levels of circulating insulin and cortisol indicate a predisposition of the animal to the development of fatty liver. Liver enzyme profile can be used to assess the extent of liver damage. Elevated ALT and AST are seen in the advanced cases of the condition. The confirmatory test is the liver biopsy or an ultrasonographic imaging of the liver.

Prevention and Treatment

Fatty liver is considered as a management related condition and efficient management of dairy animals throughout pregnancy and especially during peripartum period is required to offset this condition. Although the condition is reversible, the significance of early intervention for risk reduction and enhanced recovery cannot be understated. Dairy cows should be fed a balanced high quality diet and no sudden changes during peripartum period should be introduced. Animals should be closely monitored for their body condition score and overconditioning of animals should be

prevented. Animals should be housed in well ventilated sheds with ample provision of water to decrease their stress and maintain appetite.

Lipoptropic hepatoprotectants (methyl donors) like rumen protected methionine (RPMet), rumen protected choline (RPC), rumen protected niacin, rumen protected betaine should be added to the diet during 21 days pre and post calving period to increase the ability of the liver to export lipids in the form of VLDL as these methyl donors cause methylation of proteins which are required for the formation of lipoproteins (Arshad et al., 2023). RPC can be supplemented @ 50 gm per day (equivalent to 15 gm choline ion) from 21 days pre calving to 60 days post calving for increased hepatic export of VLDL. RPMet feeding should be increased from 8-10 gm per cow per day for the last 21 days pre partum to 15-20 gm per cow per day for the 21 days post-partum ensuring that the Lysine: Methionine content of the feed should be maintained at 2.8: 1. Monensin supplemented in the feed during dry period can reduce the risk of ketosis and fatty liver in cows by increasing the production of propionate in the rumen (Razaei et al, 2023; Mammi et al., 2021). A palatable negative dietary cation anion difference (DCAD) diet (-100 meq/ Kg feed) should be fed to the animal 21 days pre-partum for acidifying blood to reduce the risk of developing milk fever and other transition period related disorders (Santos et al., 2019).

For treatment, oral glucogenic precursors like propylene glycol @ 250 ml per cow twice a day, sodium propionate @ 200 gm per cow twice a day can be given in mild cases while more severe cases can be treated with intravenous infusion of 50 % dextrose to prevent ketogenesis and decrease lipolysis (Grunberg, 2024).

Conclusion: Fatty liver is a serious but preventable metabolic condition with a huge impact on the health and production of the animal. As the mild form of the disease is rarely diagnosed timely and herds can have an incidence rate as high as 50 %, it can cause significant economic losses to the farmer. Moreover, recovery of affected animals takes months and their susceptibility to other metabolic and infectious diseases also increases. So, appropriate dietary and management strategies for its prevention should be adopted well in time. With inclusion of rumen protected methyl donors in the high quality animal diet, preventing over conditioning in periparturient animals and ensuring a stress free environment during transition period, the condition can be smoothly circumvented.

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