Sadam D. Hasan, *BVMS, MSc, PhD*
(Lect.), Department of Internal and Preventive Medicine
College of Veterinary Medicine, University of Mosul, Mosul, Iraq
https://orcid.org/ 0000-0002-3010-501x
https://www.researchgate.net/profile/ Sadam Hasan

Fatty liver in cattle (Fat cow syndrome, hepatic lipidosis, pregnancy toxemia in cattle)

Fatty liver or hepatic lipidosis is a major metabolic disease of dairy cows in early lactation and is associated with decreased health status and reproductive perfonnance.

Etiology:

*Fatty liver is caused by the mobilization of excessive quantities of fat from body depots to the liver. It develops when the hepatic uptake of lipids exceeds the oxidation and secretion of lipids by the liver.

*Excess lipids are stored as triacylglycerol in the liver and are associated with decreased metabolic functions of the liver.

* It occurs because of a sudden demand of energy in the immediate postpartum period in well-conditioned lactating dairy cows.

* It also occurs because of a sudden deprivation of feed in fat pregnant beef cattle, and is especially severe in those bearing twins.

*The decline in dry matter intake coincides with changes in reproduction status, changes in fat mass, and metabolic changes in support of lactation, and the associated metabolic signals are likely to play an important role in intake regulation.

*These signals include nutrients, metabolites, reproductive hormones, stress hormones, leptin, insulin, gut peptides, cytokines, and neuropeptides. Body fat, especially subcutaneous fat, is mobilized and deposited primarily in liver but also in muscle and the kidneys.

Epidemiology:

Occurrence and Incidence

*Fatty infiltration of the liver is common in high-producing dairy cattle from a few weeks before and after parturition1 and is associated with several periparturient diseases and an increase in the calving-to conception interval.

*In dairy cows, fatty liver occurs primarily in the first 4 weeks after calving when up to 50% of all cows have some accumulation of triacylglycerol in the liver.

*Outbreaks of the disease have occurred in dairy herds in which up to 25% of all cows were affected, with a case-fatality rate of 90%.

*Risk Factors

Host Factors: Fatty infiltration of the liver is part of a generalized fat-mobilization syndrome that occurs in early lactation, particularly in high-yielding dairy cows, as milk production outstrips appetite and body reserves are used to meet the energy deficit.

*The deficit occurs because dietary intake cannot meet the energy requirements for the high yield. Peak yields of milk are reached 4to 7 weeks after calving, but the highest levels of voluntary feed intake are not reached until 8 to 10 weeks after calving.

*As a result of the energy deficit, the cow mobilizes body reserves for milk production and may lose a large amount of body weight.

*The disease can occur in non-lactating dairy cows by the imposition of a partial starvation diet in late pregnancy in an attempt to reduce the body weight of cows which are considered to be too fat.
*In the fatty livers of overfed cows, the rate of gluconeogenesis is not optimal, which results prolongation of lipolysis, particularly during the first few weeks after parturition. The increased lipolysis after parturition leads to a major increase in the hepatic triacylglycerol concentration and to a shift in hepatic fatty acid composition.

Pathogenesis:

*Under normal physiological conditions, the total amount of fat increases in the liver beginning a few weeks before calving, rises to an average of about 20% 1 week after calving and declines slowly to the normal level of less than 5 % by 26 weeks after calving.
*Fat mobilization begins about 2-3 weeks before calving
* mobilization of fat from body reserves, usually subcutaneous fat, to the blood that transports it to body tissues, particularly the liver but also muscle and the kidneys.
*Any decrease in energy results in the mobilization of an excessive amount of non-esterified fatty acids (NEFAs).
*This results in increased hepatic lipogenesis with accumulation of lipid in enlarged hepatocytes, depletion of liver glycogen, and inadequate transport of lipoprotein from the liver.
* lipid infiltration of the liver is in the form of triacylglycerol because of the increased uptake of NEFAs and a simultaneous increase in triacylglycerol acyltransferase.
* the loss of body condition is a result of total tissue mobilization (protein and fat) rather than fat alone.

Clinical findings:

*In dairy cattle, fat cow syndrome occurs usually within the first few days following parturition and is commonly precipitated by some conditions such as Parturient hypocalcemia, Left-sided displacement of the abomasum, Indigestion, Retained fetal membranes and Dystocia. The affected cow usually does not respond to treatment for some of these diseases and becomes anorexic
*Affected cows are usually excessively fat Excessive quantities of SC fat are palpable over the flanks, the shoulder areas and around the tail head
*Periods of prolonged recumbence are common and affected cows may have difficulty in standing when.
*The temperature, heart rate, and respiration are within normal ranges. Rumen contractions are weak or absent, and the feces are usually scant.
* A severe ketosis which does not respond to the usual treatment may occur with marked ketonuria
*Affected cows will not eat and gradually become weaker and progress to totally recumbent, and they die in 7 to 10 days.
5-Some cattle exhibit nervous signs consisting of a staring gaze, holding the head high and muscular tremors of the head and neck.
*Terminally there is coma, tachycardia, and marked hyperglycemia. The case-fatality rate in severe cases may reach 50% or more.

In fat beef cattle shortly before calving:

*affected cows are aggressive, restless, excited, and uncoordinated with a stumbling gait; sometimes have difficulty in rising; and they fall easily.
*The feces are scant and firm, and there is tachycardia. When the disease occurs 2 months before calving, the cows are depressed for 10 to 14 days and do not eat.
*Eventually they become sternally recumbent.

*The respirations are rapid, there may be an expiratory grunt, and the nasal discharge is clear, but there may be flaking of the epithelium of the muzzle.
*The disease is highly fatal; the course is 10 to 14 days, and terminally there may be coma, with cows dying quietly.

Clinical pathology:

*****Increased plasma/serum non-esterified fatty acid, acetoacetate, BHB, and total bilirubin concentrations, and decreased serum fructosamine concentration
1-High level of AST.GGT.SDH glutamate dehydrogenase, alkaline phosphatase, and gamma-glutamyl transferase activities,
2-cowsite test or Rotheras test for detection of ketone bodies
3-Liver biopsy: A liver biopsy can be used to determine the severity of the fatty liver and the concentration of triglyceride
4- Ultrasonography of liver: Ultrasonography of the liver has been used to evaluate fatty infiltration in dairy cattle

Necropsy findings:

*In severe fatal cases, the liver is grossly enlarged, pale yellow, friable, and greasy.
*Mild and moderate cases are usually not fatal unless accompanied by another fatal disease such as peracute mastitis.
* The histological changes include the occurrence of fatty cysts or lipogranulomas, enlarged hepatocytes, compression of hepatic sinusoids, a decreased volume of rough endoplasmic reticulum and evidence of mitochondrial damage.

Deferential Diagnosis:

*****Left-sided displacement of the abomasum
*Retained placenta and metritis
*Primary ketosis

Treatment:

* The prognosis for severe fatty liver is unfavorable and there is no specific therapy.
*decrease the rate of fat mobilization and therefore the plasma NEFA concentration; propylene glycol appears to act partly by this mechanism.
2- Fluid and electrolyte therapy: glucose and multiple electrolyte solutions, and the intraruminal administration of rumen juice (5-10 L) from normal cows in an attempt to stimulate the appetite of affected cows. Water and multiple electrolytes (10-30 L) can be administered intraruminal.
3-Glucagon. The subcutaneous injection of 15 mg/d of glucagon for 14 days beginning at day 8 postpartum decreases liver triglyceride concentrations in cows older than 3.5 years.
*Glucagon improves the carbohydrate status of cows by stimulating hepatic gluconeogenesis, glycogenolysis, amino acid uptake, and ureagenesis. The effect of glucagon on lipid metabolism is both direct and indirect because it directly increases lipolysis in adipose tissue but indirectly decreases lipolysis by increasing concentrations of plasma glucose and insulin.
4-Glucocorticoids. Prednisolone at 200 mg 1M or Dexamethasone-21-isonicotinate (20 to 25 mg, IM) decreases hepatic total lipid and triglyceride concentrations.
5-Propylene glycol given orally at given orally at 300 mL/ day for 5 days promotes gluconeogenesis and is used for the treatment of ketosis..
6-Insulin as zinc protamine at 200-300 SC twice daily promotes the peripheral utilization of glucose.
7-Cyanocobalamin (vitamin B12, 1 to 4 mg IV, daily for 2 to 3 treatments).

Control

*Control and prevention of fatty liver in cattle will depend on decreasing or eliminating most of the potential risk factors for the disease.

*Monensin (controlled-release capsule, 335 mg/ day) .

*Propylene glycol (300 to 500 mL daily for 5 days, PO)

*Cyanocobalamin (vitamin B12, 1 to 4 mg IV, daily for 2 to 6 treatments before or at calving