Leading Edge Veterinary Services
Hayward, WI

Ketosis

Ketosis is a complex metabolic disease that occurs when the energy requirements of an animal exceed the energy intake (Negative Energy Balance). When the body demands more energy than the diet is providing; fat deposits in the body release free fatty acids. The liver is the primary site of energy production and location where free fatty acids accumulate. In the liver, the free fatty acids are converted to energy in the form of ATP and low density lipoproteins (LDL). By products of this process are ketone bodies and fat droplets. There are three ketone bodies; acetoacetic acid, acetone and beta-hydroxybutyrate (BHB). During a state of ketosis, the accumulation of free fatty acids in the liver is too much for normal energy conversion and an excessive build up for ketone bodies occurs. Those ketone bodies are released into the blood stream and result in decreased blood pH (ketoacidosis), decreased appetite and eventual organ damage secondary to acidotic state.

There is another disease that goes hand in hand with ketosis; this disease is hepatic lipidosis or fatty liver disease. Fatty liver disease occurs when excessive amounts of fat droplets are deposited into the liver. This occurs when there are excessive amounts of free fatty acids accumulating in the liver secondary to a ketotic state. Excessive fat droplets in the liver damage liver cells. The damaged liver cells are not as efficient at producing energy (ATP and LDL), thus perpetuating the cycle. Eventually liver failure occurs when enough liver cells are damaged.

Type One: Peak Lactation Ketosis

- Occurs 3-6 weeks into lactation.
- Diet is not meeting energy needs for high production
- Weight loss and tends not to be associated with severe fatty liver infiltration

Type Two:

- Occurs 1-2 weeks into lactation
- Thought that increased adipose sensitivity and insulin resistance, occurring in the periparturient period, results in increased lipolytic response to a given stimulus (low blood glucose)
- Ketosis is associated with high NEFA mobilization and greater fatty liver infiltration.

Butyric Acid-Induced Ketosis

- Caused by the direct consumption of ketones in the diet.
- Results in poor dry matter intake
- Similar clinical syndrome as type one ketosis.

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Treatment

- Eliminate negative energy balance and the factors causing it. Treat any existing primary disease. Offer highly palatable, highly digestible ration.
- 250ml 50% Dextrose IV
- 8 ounces of Propylene glycol by mouth once daily for 2-4 days.
- Glucocorticoids once; stimulates gluconeogenesis (enhance mobilization of glucose precursors (amino acids) and temporarily decreases milk production (reduces glucose uptake by mammary gland) and stimulates appetite.
- B-vitamins. Cofactor in TCA cycle.

Prevention

- Prevent obesity in late lactation/dry period. Goal should be a dry off and maintain body condition score at 3.25 – 3.75
- Feed palatable, highly digestible feeds late gestion and early lactation.
- Increase level of grain feeding 3-4 weeks prepartum to adapt rumen microbes so about to absorb VFA.
- Good feeding management to maximize feed intake.