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Popular Article

An overview of Ketosis in cows

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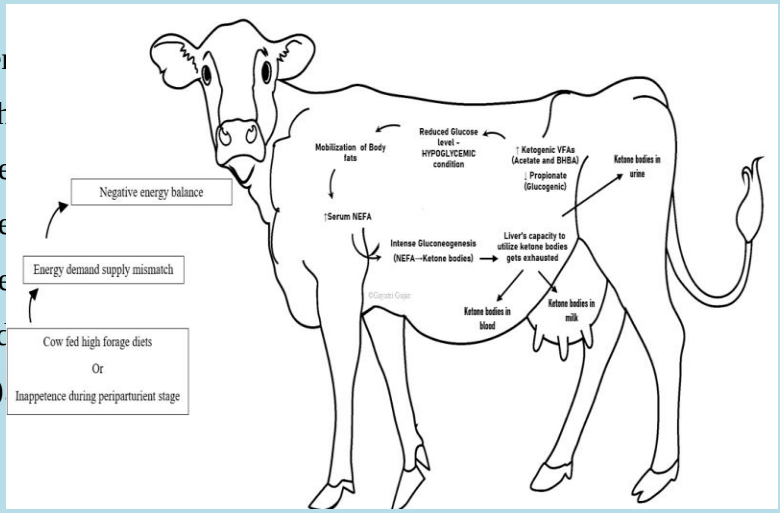
Introduction

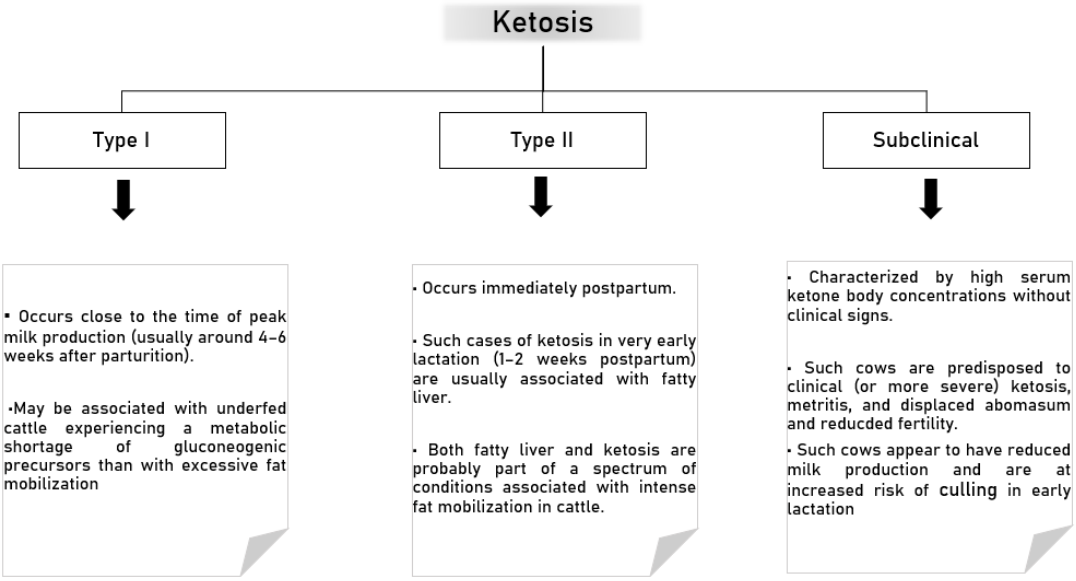
Ketosis literally translates to the "elevated concentration of ketone bodies viz., acetone, acetoacetate, beta-hydroxybutyrate in all body fluids, commonly manifested as anorexia, decreased milk production, evident loss of body condition, solid dry faeces, and, in rare conditions with neurologic signs (nervous ketosis). However, most of the time ketosis may go undiagnosed and remain subclinical. Ketosis is a common disease of adult cattle. It typically occurs in dairy cows in early lactation and is most consistently characterized by hyporexia and reduced milk production. Rarely, it occurs in cattle in late gestation, at which time it resembles pregnancy toxemia of ewes. In addition to inappetence, clinical signs of nervous dysfunction, including pica, abnormal licking, incoordination and abnormal gait, bellowing, and aggression, occasionally occur. Ketosis is worldwide in distribution; however, it is most common where dairy cows are bred and managed for high production. Cows with ketosis can be identified via routine testing using appropriate cowside blood, milk, or urine tests. The most efficacious treatment for ketosis is oral drenching of propylene glycol.

Etiology And Pathogenesis of Ketosis in Cattle

In contrast to many other species, cattle with hyperketonemia do not have concurrent acidemia. The serum ketone bodies are acetone, acetoacetate, and beta-hydroxybutyrate (BHB)



Types of Ketosis



In practical terms, differentiating clinical and subclinical ketosis is insignificant and may be difficult to determine without routine testing. Determination of serum or whole blood BHB concentration is considered the best way to detect and monitor subclinical ketosis; however, urine or milk cowside tests can also be used in on-farm monitoring programs. Concentrations >1.0 mmol/L (10.4 mg/dL) or 1.4 mmol/L (14.6 mg/dL) blood or serum BHB are considered diagnostic of subclinical ketosis. The standard threshold used for blood is 1.2 mmol/L (12.5 mg/dL), which corresponds to thresholds of 100 mcmol/L for milk, and 15 mg/dL (or "small" on a dipstick) for urine.

Epidemiology Of Ketosis in Cattle

- Dairy cows in early lactation (the first 6 weeks postpartum) are particularly vulnerable, with most cases occurring in the first 2 weeks of lactation.
- Ketosis can occur in all parties, however, risk increases with increased number of parity.
- Recent evidences like specific genetic markers found associated with ketosis risk suggest moderate heritability, and a genetic predisposition to some extent.
- Cows with excessive adipose stores (body condition score ≥3.75 on 5-point scale) at calving are at a greater risk of ketosis than those with lower body condition scores.
- Lactating cows with subclinical ketosis are also at a greater risk of developing clinical ketosis and displaced abomasum than cows with lower serum BHB concentrations.

Clinical Findings of Ketosis In Cattle

✓ Reduced dry matter intake

✓ Dehydration

✓ Reduced milk production

✓ Variable ruminal motility- being hyperactive in some cases and hypoactive in others

In many cases, there are no other physical abnormalities.

✓ In some cases, CNS disturbances are evident (nervous ketosis). Neurologic signs include abnormal licking and chewing, and sometimes incessant chewing on pipes and other objects (pica). Incoordination and gait abnormalities occasionally occur, as do aggression and bellowing. These clinical signs occur in a minority of cases, but because the disease is so common, encountering patients with these clinical signs is not unusual.

Diagnosis Of Ketosis in Cattle

Treatment Of Ketosis in Cattle

Treatment of ketosis is aimed at re-establishing normoglycemia and reducing serum ketone body concentrations.

- Propylene glycol acts as a glucose precursor, and oral drenching (250–400 g [8–14 oz], PO, every 24 hours for 3–5 days) is effective as a ketosis treatment. Overdosing propylene glycol leads to CNS depression.
- Bolus glucose treatment (500 mL of 50% dextrose solution, IV, as a single bolus) is also common. This solution is very hyperosmotic and, if administered perivascularly, results in severe tissue swelling and irritation, so care should be taken to ensure that it is given IV. Bolus glucose treatment generally results in a rapid temporary recovery, especially in cases occurring near peak lactation (type I ketosis). Additional therapy with bolus glucose treatment (500 mL of 50% dextrose solution, IV, as a single bolus) in neurologic cases and vitamin B_{12} (1.25 mg, IM, every 24 hours for 3 days) in cases that are also hypoglycemic is suggested.
- Dextrose administration is recommended for cases of nervous ketosis, but may not be necessary or even helpful for every ketosis case. Administration of glucocorticoids is not recommended as there is little evidence of benefit and some indication of harm.
- There is also support for the use of vitamin B_{12} (1.25 mg, IM, every 24 hours for 3 days) as an adjunct treatment with oral drenching of propylene glycol, particularly in ketotic cows that are also hypoglycemic.

Prevention Of Ketosis in Cattle

Encouraging greater DMI

Encouraging greater DMI is the single most effective preventative measure against ketosis. Greater DMI enables similar energy intake, at a lower energy density, which allows the necessary effective fiber levels to maintain rumen function and metabolic processes

Prophylactic treatment

Continual monitoring and early response to sub-clinical ketosis make prophylactic propylene glycol treatment a successful strategy to reduce the instances of clinical ketosis. The concept is to provide a "shock" amount of easily metabolizable glycerol that will increase circulating insulin levels sufficiently to reduce the mobilization of body fat and ease the metabolic pressure on the liver. Alternatively, propylene glycol treatment is often applied at the first sign of clinical ketosis. At this stage, damage to the liver has already been incurred and the risk of developing secondary metabolic disorders significantly increased. If clinical ketosis does occur, glucose or glucose-derivatives can be given intravenously. Most cows will respond to intravenous treatment, however, damage to the liver has likely already occurred and lactation performance will have been compromised.

Feed additives

Feed additives that encourage greater DMI will help to reduce the instances of sub-clinical ketosis. Yeast supplements are commonly fed in dairy rations for their general effect of enhancing rumen buffering capacity and improving the degradation of feeds. This action stabilizes the rumen pH and microbiota, which in turn encourages greater mobility of the cow and more time spent at the feed bunk. Greater digestion of feedstuffs increases the flow of volatile fatty acids available for glucose production. To alleviate excess fat deposition in the liver, choline and methionine can be fed to help mobilize accumulated fat out of the liver where it can then be excreted into the milk. Use of plant secondary metabolites, or phytogenic feed additives are a more natural way of encouraging greater intake by stimulating rumination and chewing duration, both of which increase absorption of rumen volatile fatty acids as well as buffering the rumen.

References

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