Ketosis (Hyperketonemia): How Should We Manage This Disease?

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At a recent session of Large Animal (LA) Rounds in our veterinary practice, the clinical condition known as ketosis was discussed by our veterinary staff. During the meeting, there was a great deal of discussion and debate regarding the treatment and management of this disease. Ketosis research is very active and presently there is no absolute consensus within the research community regarding the treatment of ketosis. However there is general agreement. Based on the recent research findings, we have refined our ketosis detection, treatment and management protocols.

Ketosis occurs when **BLOOD** betahydroxybutyrate **(BHB)** levels exceed 1.2 mmol/litre. There are three forms of the disease: *clinical*, subclinical and *nervous* ketosis. The classic case of ***clinical*** ketosis (these cows are noticeably sick with clinical signs that include reduced milk production, normal temperature, stiffer than normal manure, are bright and alert with normal hydration; still eat long stem forages well but their intake of silages, grains and pelleted feed is much reduced) generally occurs 3 to 6 weeks post-calving in high producing cows. Their BHB levels exceed 2.9 mmol/litre. They are in a negative energy balance because their energy intake can't keep up to their milk production. They are typically thin cows at the time of diagnosis because they have stripped off much of their body fat to produce additional energy for milk production. *This form of ketosis has also been called **Type 1** ketosis (later onset, usually clinical).*

The classic case of ***subclinical*** ketosis most often occurs in the first 2 weeks of lactation, usually in well conditioned or over-conditioned fresh cows. Their BHB levels are typically between 1.2 – 2.9 mmol/litre. They are subclinical meaning that they are not showing any obvious signs of illness. If not detected and treated promptly and appropriately, they often go on to develop other clinical conditions such as displaced abomasums (DAs) and metritis, and generally have poorer reproductive performance. *This form of ketosis is sometimes referred to as Type 2 ketosis (earlier onset, usually subclinica*l). In most herds, subclinical cases account for 80% of all cases of ketosis.

Nervous ketosis is a much less common form of ketosis. In addition to symptoms seen with clinical ketosis, these cows exhibit signs of nervous system derangement such as bellowing, compulsive licking or sucking, star-gazing, or incoordination. All nervous ketosis cases have BHB levels >2.9.

In addition to leading to diseases such as retained placenta, displaced abomasums (DAs) and metritis, ketosis can occur secondarily to these disease conditions and others such as hardware disease, acute mastitis, and pneumonia. The ketosis will exacerbate the primary condition. In our veterinary practice, all sick cows in the first 6 weeks of lactation are tested for ketosis. If one of these other diseases is identified, and the cow also has elevated levels of BHB, both diseases must be treated and managed to maximize the speed and degree of recovery.

Identifying clinical cases is easy by simply testing all sick cows. Identifying subclinical cases is more challenging and requires a systematic approach to testing all fresh cows and all early lactation sick cows. Many dairy herds have now implemented a Fresh Cow Monitoring program on their farms in which they check for ketosis, among other things. Research has shown that the most efficient ketosis detection protocol involves testing each fresh cow twice between 3 and 9 days in milk (DIM). This is because the highest prevalence of ketosis typically occurs between 3-9 DIM. However, following this testing protocol, you will miss approximately 25% of ketosis cases because roughly 25% of cases occur after 9 DIM. Therefore, we recommend testing early lactation cows twice, the first time between 3-10 DIM and the second time between 11-18 DIM.

Research has also clearly shown that the most accurate testing method is to measure ***blood BHB*** levels. At present, the commercially available cow-side ketosis testing methods for **milk** and **urine** (Ketotest, Ketocheck, Ketostix, and PortaBHB) are all very good at identifying a clinically ketotic cow as ketotic, but also incorrectly identify about half of subclinically ketotic cows as **not** ketotic. Therefore using these tests, many cows that could have benefited from ketosis treatment do not get treated. Research has shown that the average case of ketosis costs $290 (U.S.), and that 88% of DA cases are attributable to ketosis, and that 70% of metritis cases are attributable to ketosis. It is a very costly disease.

Consequently, since LA Rounds, our veterinarians have renewed their resolve to have all of our dairy clients on a fresh cow ketosis monitoring program, and to test all sick fresh cows, using the Precision Xtra Meter to measure **BLOOD BHB** levels (it is the same meter that human diabetics use to monitor their blood glucose levels). The meter is relatively inexpensive, is durable and easy to use. The meter, as well as the BHB test strips can be purchased at most drugstores. They do not function well in the cold weather, so keeping the meter warm is important. Blood collection with some practice, is quite easy. If you take the blood sample from the mammary vein (milk vein), the readings are 0.3 units lower than from the tail or jugular vein, so you must adjust the values accordingly.

Ketoscreen, the CanWest DHI ketosis monitoring service, is a very useful herd monitoring tool to provide producers an indication of the herd prevalence of ketosis. If you do not presently have a Ketosis monitoring program on your farm for fresh cows, we highly recommend Ketoscreen as a place to start. Research has shown that on the average dairy farm, between 20 and 40% of cows in the first 6 weeks of lactation have ketosis.

Since LA Rounds, the treatment protocols are based on the level of blood BHB found:

Blood BHB>1.2 and <3.0 (Subclinical Ketosis: first 2 weeks of lactation, no clinical signs)

1. Administer propylene glycol (PG) orally at the label dose 300 grams (500-600 millilitres of a 67% PG solution like Glycol P) once daily for 3 to 5 days
2. 1500 micrograms of Vitamin B12 intramuscularly once daily for 3-5 days
3. Do **NOT** administer intravenous Dextrose to these cows, particularly if they are in the first 2 weeks of lactation. The Dextrose could worsen their illness because these cows often already have high blood glucose levels.
4. If the cow has not yet received a Rumensin bolus pre-calving or post-calving (and is either not receiving Rumensin in the feed as a premix, or her daily dose from the premix is less than 350 mg), administer a Rumensin bolus. If she is receiving a significantly higher daily dose of Rumensin from a premix, be cautious about administering a Rumensin bolus.

Blood >2.9 (Clinical Ketosis and Nervous Ketosis: 3-6 weeks of lactation)

Treat as per Subclinical Ketosis**, PLUS**:

1. Administer PG for a minimum of 5 days
2. 250 ml of a 50% Dextrose solution intravenously (never more than 250 ml, never more than once)
   ****Administering Dextrose to these cows is controversial****

3. If you have your own stomach pumping system (or if your veterinarian is treating the cow and has one), orally administer a glucose precursor such as calcium propionate. One pound of calcium propionate is recommended. We administer it in 20-40 litres of warm water.

In all cases of ketosis (in particular subclinical cases), we recommend that the cow is retested for blood BHB levels on the last day of treatment of PG. If her BHB level is still greater than 1.2, she must be re-examined, preferably by your veterinarian to determine why she has not responded. The treatment protocols listed above are very effective in the treatment of ketosis. In most cases, another clinical condition will be identified that was not present when treatment was initiated.

Infrequently, in herds that routinely administer Rumensin boluses to their pre-fresh cows (more so before Elanco put a stronger spring in their Rumensin boluses), we would get relapses 2-5 days after the end of treatment with PG and Vitamin B12. When many of these cows were re-examined, no other medical condition could be identified to explain the relapse. We postulated that either the cow had expelled the Rumensin bolus or it had become plugged with feed. In these cases, we administer a second Rumensin bolus. In my experience, when these cows received a second bolus, the relapses stop.

Prevention of ketosis involves the appropriate management, feeding and housing of transition dairy cows. We are big advocates of Rumensin as a premix in the feed or as a bolus to aid in the prevention of ketosis. However, we never administer a Rumensin bolus to Ayrshire cattle due to their inherent sensitivity to Rumensin.

This article outlines the opinions of our veterinary practice at this date and time, and our opinions may change as further research findings become available. These opinions may not be consistent with the opinions of your herd veterinarian. Please consult **your** herd veterinarian regarding the management, treatment and control of ketosis on **your** farm.