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METABOLIC HEALTH IN DAIRY CATTLE: A PRACTICAL GUIDE TO PREVENTION, MANAGEMENT AND TREATMENT

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Abstract

Metabolic disorders are major health challenges in dairy cattle, particularly during the transition period, which extends from approximately three weeks before to three weeks after calving. During this period, increased nutrient demands for fetal growth and milk production, coupled with inadequate feed intake and hormonal changes, can disrupt energy and mineral metabolism. These disturbances may lead to ketosis, fatty liver, hypocalcemia and subacute ruminal acidosis, adversely affecting milk production, reproductive performance, immune function and animal welfare. Both clinical and subclinical disorders can cause substantial economic losses to dairy farmers. Effective prevention requires appropriate body condition management, balanced transition diets, adequate mineral supplementation, gradual adaptation to lactation rations and regular monitoring of metabolic health. Early diagnosis and timely, condition-specific treatment

can further reduce complications and improve recovery. Effective prevention and management of metabolic disorders in dairy cattle through proper nutrition, regular health monitoring, and appropriate transition-cow management are essential for reducing disease incidence, improving reproductive performance, maintaining milk production, and ensuring the health and productivity of dairy herds.

Keywords: Dairy cattle, metabolic disorders, transition period, ketosis, hypocalcemia, fatty liver, prevention, herd health.

Introduction

Metabolic disorders are major health and production challenges in dairy cattle, particularly during the transition period (three weeks before to three weeks after calving). Increased nutrient demands and inadequate feed intake can predispose cows to ketosis, fatty liver, hypocalcaemia and ruminal acidosis. Negative energy balance promotes fat mobilisation, increasing circulating non-esterified fatty acids (NEFA) and the risk of ketosis and fatty liver. These disorders can reduce milk production, impair reproductive performance and compromise animal welfare. Subclinical conditions are especially concerning because they may remain undetected while affecting productivity. Effective prevention requires balanced nutrition, appropriate body condition, adequate mineral supply and close monitoring around calving. Early diagnosis and timely veterinary intervention are essential for improving herd health, productivity and farm profitability.

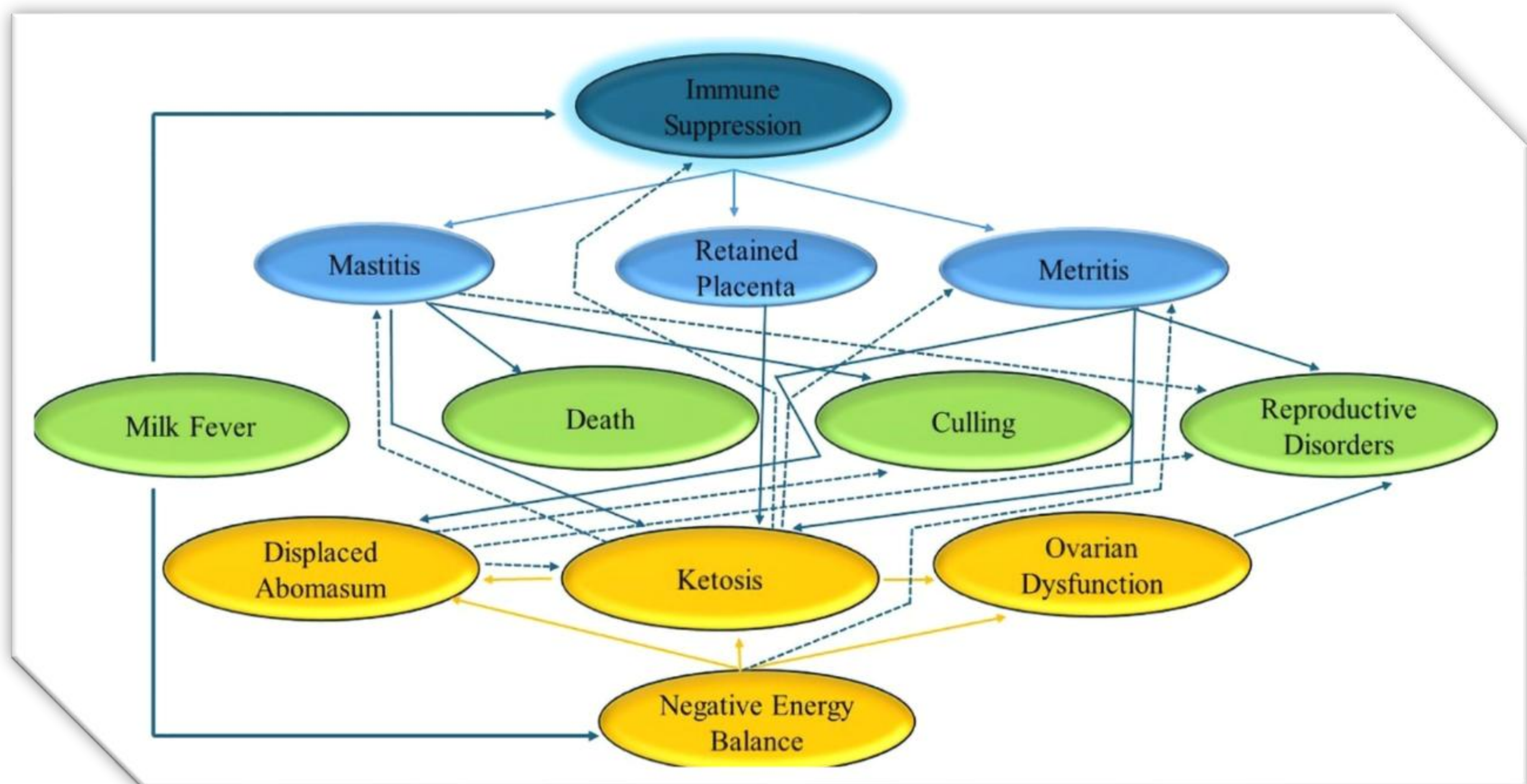


Figure 1. Interrelationships among metabolic disorders, immune suppression, and health complications in transition dairy cows.



Metabolic profile testing: A herd-health approach

Metabolic disease prevention should move from treating individual cows to monitoring the herd. Depending on the clinical problem, useful indicators include blood calcium, magnesium and phosphorus; BHB and NEFA for energy metabolism; glucose; urea; albumin and total protein; liver enzymes; and selected trace minerals. Sampling should be timed according to the transition stage and interpreted with the ration, production records and clinical findings. For ketosis, BHB is particularly useful for detecting subclinical disease. For hypocalcaemia, sampling around calving can help evaluate the success of a transition-cow program. Laboratory results should never be interpreted in isolation.

Milk fever (Parturient paresis / Hypocalcaemia):

It is an acute disorder of calcium homeostasis occurring most commonly around calving. It is an acute calcium-deficiency disorder and emphasizes pre-calving dietary management, acidogenic rations and calcium supplementation. Early signs may include restlessness, reduced appetite, weakness, cold extremities and muscle tremors. Affected cows may become sternal and then lateral recumbent. Severe cases may show depressed consciousness, flaccid paralysis, coma and death.

Prevention Maintain appropriate body condition and avoid over-conditioning during the dry period. Use a properly formulated close-up ration. Anionic/acidogenic diets are an evidence-based strategy for high-risk dairy cows. Control dietary potassium when appropriate because high potassium intake can impair calcium homeostasis. Use oral calcium supplementation strategically in high-risk cows. Monitor transition-cow performance and, in herd programs, evaluate calcium status in representative cows.

Treatment

Primary Intervention: Administer 300-600 ml of a 25% calcium borogluconate solution intravenously (IN).

Prevent Relapse: Follow IN administration with an intramuscular (IM) or subcutaneous (SC) injection for slow, sustained release and prevention of heart block (e.g., Thiacal 450 ml).

For Complicated Cases: Use composite solutions containing Ca, Mg, P, and glucose.

Oral Supplementation: Administer calcium gel orally one day before and one day after calving.

Prophylactic Vitamin D: Administer Vitamin D or 1,25-dihydroxyvitamin D subcutaneously 8 days before calving.

Downer cow syndrome:

Downer cow syndrome describes prolonged recumbency in a cow unable to rise. It is a clinical syndrome rather than a single disease. Milk fever is an important cause, but nerve injury, muscle damage, calving trauma, hypophosphataemia, hypomagnesaemia and other metabolic or musculoskeletal conditions must also be considered. The uploaded presentation highlights prevention of milk fever, body-condition management, comfortable bedding and supportive care.

Management principles

- Correct specific metabolic abnormalities such as hypocalcaemia, hypomagnesaemia or hypophosphataemia when confirmed or strongly suspected.
- Provide deep, dry, non-slip bedding, adequate space and easy access to water and palatable feed.
- Reposition the cow regularly and protect pressure points, avoid repeated traumatic attempts to force the cow to stand; lifting aids should be used appropriately and only when indicated.

Core Treatment

- **Initiate Calcium Therapy:** Administer calcium as the first line of treatment.
- **Provide Broad Spectrum Mineral Support:** Give additional treatment with Potassium (K), Phosphorus (P), and Magnesium (Mg).
- **Monitor Blood Mineral Status:** Regularly check blood levels to guide ongoing therapy.

Ketosis:

It is a disorder of energy metabolism, most common in early lactation, in which negative energy balance and excessive fat mobilization lead to increased ketone bodies. When glucose supply does not meet lactation requirements, adipose tissue releases fatty acids. The liver takes up non-esterified fatty acids; when hepatic processing capacity is exceeded, ketogenesis increases. Beta-hydroxybutyrate (BHB) is an important measurable ketone body. Obesity at calving and excessive body-condition loss increase risk.

Prevention

- Prevent cows from becoming excessively fat before calving. Maximize dry-matter intake during the transition period through comfortable housing and palatable, well-formulated rations. Provide adequate effective fibre while avoiding abrupt dietary changes.

- Administer niacin 6-8 gm/cow/day orally, starting 20 days pre-calving and continuing for up to 3 months post-parturition.
- Provide Sodium Propionate 100 gm daily for 6 weeks as a targeted herd-level intervention.

Treatment

1. Glucogenic Precursors

- **Propylene Glycol (Cows):** 250 ml mixed with an equal amount of water, given orally per day. *Mechanism: Increases glucose, reduces insulin, and reduces fat mobilization.* **Glycerol:** 100mg twice daily for 3 days. *Mechanism: Aids in gluconeogenesis.*

2. Hormonal & Vitamin Therapy

- **Dexamethasone:** 30 mg IM injection. Mechanism: Reduces ketone body formation, **Niacin:** 6-8 gm/day for 1-2 weeks. Mechanism: May reduce blood ketones, **Insulin:** 200 IU. Mechanism: Facilitates glucose transport into cells.

Hypomagnesaemic tetany (Grass tetany):

It is an acute, potentially fatal disorder associated with low magnesium availability, classically affecting cattle grazing lush, rapidly growing pasture. Risk factors are lush spring pasture and rapidly growing green forage, High potassium and nitrogen fertilization, Low magnesium intake or poor magnesium availability and High-producing lactating cows with increased mineral requirements.

Prevention Strategies:

- **Manage Pasture Access:** Prevent cows from grazing exclusively on excessive green, lush pastures. **Avoid High N & K Fertilizers:** Do not use fertilizers high in Nitrogen and Potassium on pastures, as they interfere with Mg absorption. **Provide Free-Choice Mineral Mixes:** Offer a mix of MgO and salt (75:25 ratio) ad-lib. Provide a salt lick with 10 parts MgSO₄, 10 parts Ca diphosphate, and 80 parts salt.
- **Ensure Daily Magnesium Intake:**
 - o Provide 10-15 g Mg/day for pregnant cows and 30 g/day for lactating cows.
 - o Mix 60 g of magnesium oxide into 0.5-1 kg of grain concentrate for effective delivery.

Clinical signs and Treatment

Affected cattle may show nervousness, muscle tremors, hyperexcitability, staggering, collapse, convulsions and death. Treatment is an emergency. Calcium and magnesium-containing injectable solutions are commonly used under veterinary supervision. The animal should be removed from the high-risk pasture and provided with an appropriate magnesium source after stabilization. Because sudden death can occur, prevention is more effective than attempting to treat advanced grass tetany.

- **Administer Calcium & Magnesium Solution:** Use a combined solution (e.g., Mifex), giving half I/V and half S/C.
- **Provide Subcutaneous Magnesium Sulphate:** Administer 200 ml of a 50% solution of Epsom salt (MgSO₄) S/C.
- **Calf Protocol:** Administer 10 mg MgSO₄ in sterile water S/C.

Post-parturient haemoglobinuria (PPH):

It is an uncommon but potentially serious disorder affecting high-yielding dairy cows during early lactation. It is characterised by intravascular haemolysis, anaemia and haemoglobinuria. Hypophosphataemia is an important associated factor, although copper deficiency and certain dietary factors may also contribute. Clinical signs include reduced milk yield, anorexia, weakness, pale or yellowish mucous membranes, increased heart and respiratory rates, and dark red or brown urine. Severe anaemia can be life-threatening; therefore, prompt veterinary diagnosis and appropriate treatment are essential.

Immediate Correction of Anaemia

- **Blood Transfusion:** Transfuse up to 5 liters of whole blood.

Specific Therapy to Correct Hypophosphatemia

- **Intravenous Phosphorus:** Slowly administer 80 g of Sodium acid phosphate dissolved in 400 ml of distilled water I/V.
- **Injectable Phosphorus:** Administer Tonophosphan injection.
- **Oral Phosphorus:** Provide oral dosing with bone meal (120 gm twice daily).
- **Ascorbic Acid:** Administer 5 gm per cow I/V.

Supportive Therapy: Fluid Therapy: Administer fluids to minimize the danger of haemoglobinuric nephrosis (kidney damage). **Haematinic Drugs:** Use preparations containing Copper (Cu), Cobalt (Co), and Iron (Fe). **Liver Tonics:** Administer 20-30 ml of a liver tonic (e.g., vetliv, livogen) orally

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