MILK FEVER

(HYPOCALCAEMIA, PARTURIENT PARESIS, ECLAMPSIA)

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OVERVIEW

- average annual incidence in dairy herds approximately 5%
- Individual prevalence of 60–70% when calving at pasture
- especially dairy crosses (e.g. Hereford × Holstein)
- more common in autumn-calving cows

AETIOLOGY

- Blood calcium is kept under homeostatic control via:
  - parathyroid hormone (PTH)
  - 1,25-dihydroxy cholecalciferol (calcitriol)
  - Calcitonin
- Sudden production of 40 liters milk → extra dietary intake of 80 g of calcium/day
- Ca: absorption ↑ - mobilization from skeleton (2-3 days)

Predisposing Factors

- Age: Older cows respond more slowly and are thus more prone to milk fever (3rd to 7th parturition)
- Magnesium status
  - Mg $\downarrow \longrightarrow$ may interfere with release of PTH
  - Mg $\downarrow \longrightarrow$ activity of PTH on its target tissues & hydroxylation of vitamin D3 in the liver
- Ca dietary level in dry period
  - Ca $\downarrow \longrightarrow$ PTH $\longrightarrow$ mobilization of Ca from bones / absorption from gut

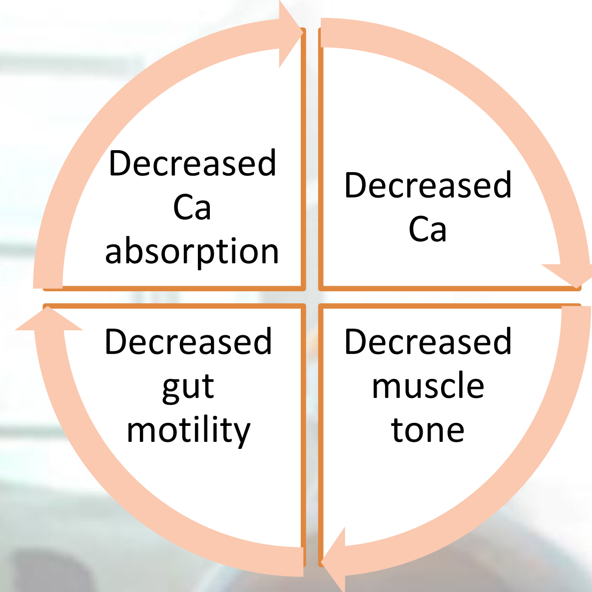
Predisposing Factors

- Acid–base status
  - Metabolic alkalosis affects Ca metabolism ⇒ Milk Fever
- Nutritional factors
  - Decreased DM intakes in cows at calving ⇒ ↓ Ca absorp.
  - Digestive upsets (e.g. diarrhea) ⇒ ↓ Ca absorption
- Breed

Channel Island breeds (esp. Jerseys) > Holstein Friesians

(age should be considered)

Pathophysiology



- Calcium

✓ Increase cell membrane stability

✓ Necessary for Ach release in nerve-muscle terminal

✓ Essential for muscle contraction

✓ Builds and maintains bones and teeth

✓ Assists in normal blood clotting

✓ Calcium is also essential for optimum milk production and growth rate.

CLINICAL PRESENTATION

- Usually occur within 24-48 hours after parturition
- can occur at or before calving
- Exceptionally during estrus in very high-yielding cows (several weeks to months after calving)
- Progressive clinical signs over 12-24 h
- ***Initial signs (stage 1):*** hyperaesthesia, teeth grinding and coarse muscle tremors, stiffness of the limbs, straight hocks and ‘paddling’ of the feet when standing, ataxia &reluctance to walk, normal or slightly depressed rectal temperature

CLINICAL PRESENTATION

Clinical signs of hypocalcaemia usually occur within 24 hours after parturition, as in this beef cow. Note the alert calf, but depressed dam

CLINICAL PRESENTATION

- ***Clinical signs progress (stage 2):*** sternal recumbency with a lateral kink (‘S-bend’) in the neck, then the head becomes averted against the chest, increased heart rate, dilated pupils (with reduced papillary light reflex [PLR]), gut stasis causing bloat and constipation, and profound depression
- ***More progress (stage 3):*** comatose in lateral recumbency, very weak, elevated heart rate (120 beats per minute), ruminal tympani and/or paralysis of respiratory muscles causes death in untreated cattle
- ***Potential Complications***: uterine inertia (leading to dystocia and/or stillbirth &/or RFM), prolapse of the uterus and musculoskeletal damage, metritis and endometritis, thus affecting subsequent fertility. Downer cow or trauma in late treatment

head being averted against the chest

sternal recumbency, with a lateral kink (‘S-bend’) in the neck

CLINICAL PRESENTATION

This cow with hypocalcaemia has become comatose in lateral recumbency during the latter stages. Note the associated dystocia because of uterine inertia.

DIFFERENTIAL DIAGNOSIS

- Acute toxic mastitis
- Physical injury/nerve paralysis
- Uterine rupture
- Hemorrhage caused by dystocia
- Acidosis
- Grain overload

DIAGNOSIS

- Clinical
- The cow's history
- Clinical signs
- Response to intravenous calcium borogluconate solution within minutes
- Laboratory
- Ca levels fall to <1.5 mmol/l or <5 mg/dl (normal 2.2–2.6 mmol/l; 8.5-10.4 mg/dl)
- Phosphate↓ (<1.0 mmol/l; normal range 1.4–2.5 mmol/l) often observed (will be restored by Ca treatment)
- Mg levels: slight hyper-magnesaemia (>1.25 mmol/l)
- Glucose : 95-135 g/dl
- CPK & AST level increased

MANAGEMENT

- Previous **SC** Ca inj by owner (slow absorption of 3-4 hours if any; due to weak circulation)
- IV ***infusion*** of Ca borogluconate solution in 5 -10 min (40% solution, 400 ml; 1 g/45 kg BW)
- SC inj. of Ca for ***slow*** release of Ca for high yielding cow may be necessary (25-30% recurrence)
- Note: Ca is toxic for heart, control heart function
- SC or Oral (Calcium propionate) administration of Ca in mild cases
- Dairy cows should not be milked for 24 hours and the calf removed after feeding colostrum

Sudden death may also occur after

Ca injections if cow excited or frightened (increased sensitivity to EP)

Exposure to sun or a hot, humid atmosphere, heatstroke may be a ***complicating factor***

- Recumbent cows must be kept in sternal recumbency (not in lateral recumbency), to avoid regurgitation and aspiration pneumonia
- Roll the cow from side to side every few hours
- Provided with adequate bedding or moved to a suitable non -slip ground surface

Sings of response to calcium therapy…

1-Belching

2- Muscle tremor, particularly of the flanks and often extending to the whole body

3- improvement in the amplitude and pressures of the pulse

4-Increase in the intensity of the heart sounds

5-Sweating of the muzzle

6- Defecation.

Prognosis

- The longer the period from onset of milk fever to treatment, the longer the period of post-treatment recumbency and the higher the case-fatality rate

Prevention/control measures

- ***Over fatness*** in dry cows should be avoided: the aim should be calve cows at body condition scores of 2.5–3.0 out of 5.
- Manipulation of the dry cow diet is the most cost-effective method of controlling the incidence of hypocalcaemia:

1. The amount of ***calcium*** in the transition dry cow diet should be limited to ‹50 g/head/day (ideally ‹30 g/day) to maintain PTH activity. ***Magnesium*** levels in the diet should be ›40 g/day (very difficult in grass-based forage systems due to the relatively high levels of calcium in grass. The use of calcium-binding agents such as zeolite may help reduce calcium absorption).
2. Manipulation of the DCAB, which by ***lowering blood pH*** allows PTH to act on its receptor and releases cations (mainly calcium) from bone. Sodium, potassium, sulphate and chloride ions exert the strongest effects on acid–base balance, and are referred to as the strong ions

Prevention/control measures

- ***Calcium by mouth*** at calving. Cows known to be at risk of milk fever can be given calcium at/just before calving using drenches (150 g calcium chloride {metabolic acidosis} or Ca propionate daily), gels and calcium boluses.
- Administration of 250 mg ***vitamin D3*** (cholecalciferol) or alfacalcidol (1α-hydroxycholecalciferol) can be carried out prior to calving, but this necessitates accurate prediction of calving date.

SHEEP

- ***outbreaks*** in groups of ewes: forced exercise, long-distance transport, sudden deprivation of food and grazing on oxalate-containing plants or green cereal crops
- mature ewes are the most susceptible; young ewes up to one year when graze green oats &/or pasture is short
- 6 weeks before to 10 weeks after lambing
- Up to 25 % of the flock

SHEEP

- Clinical manifestation: paresis; in the rest of the flock poor growth, lameness and bone fragility can be detected
- Incidence rate: 10%
- Fatality rate: 20%
- Hypocalcemia in sheep depresses endogenous glucose production and in late pregnancy in combination with hyperketonemia, facilitates the development of pregnancy toxemia

GOAT

- Milking goats become affected mostly during the ***4-6-year age*** group
- Before and after kidding (some later than 3 weeks after parturition)
- ***Clinical syndromes*** are identical to those in cows, including the two stages of ataxia and recumbency
- Serum calcium levels are reduced from normal levels for parturient does of 9.4-3.6 mg/dL (2.35-0.9 mmol/L)

DOG: Eclampsia (hypocalcemia)

- Small breed dogs
- Large litter size
- 2-3 weeks after parturition (nursing pups)
- Ca supplements during pregnancy can be a cause
- ***Clinical signs:*** 1-restlessness, muscle weakness or tremors, facial pruritus, and neglect of the pups. 2-Tachycardia or bradycardia, and midriasis. 3-End-stage eclampsia is characterized by convulsions and subsequent hyperthermia.
- ***Management:***

1. Cool the body
2. Diazepam for convulsions
3. IV infusion of Ca gluconate (22-44 mg/kg)
4. Oral supplementation of Ca (1-3g/day) + Vit. D (10000-25000iu) to avoid recurrence

Horse: Eclampsia (hypocalcemia)

- characterized by abnormal behavior such as incoordination and tetany
- predisposing factors: High-producing mares; physical hard work; grazing on lush pasture; Diet high in Ca in late pregnancy; sudden change in diet after parturition

Clinical sings ….

- 1-affected animals sweat profusely and have difficulty in moving because of tetany of the limbs and incoordination. The gait is stiff and the tail is slightly raised. Rapid, labored respirations and wide dilatation of the nostrils are often accompanied by diaphragmatic flutter(‘thumps')or thumping sound
- 2- Muscular fibrillation, particularly of the masseter and shoulder region ,and trismus are evident but there is no prolapse of the 3rd eye lid.
- 3- Within about 24 h the untreated mare becomes recumbent, tetanic convulsions develop and die with in about 48 h after the onset of illness.

Horse: Eclampsia (hypocalcemia)

- D.D… 1-Tetanus 2-Laminitis
- Treatment…. 300-500 mL of a 25% solution of calcium borogluconate or gluconate administered slowly IV (over 15-30 min) is recommended .

Subclinical hypocalcaemia

- (total plasma calcium <1.9 mmol/L) occurs in dairy cattle during the first few weeks of lactation.
- Incidence based on Ca levels in blood 23% to 39% (Up to 33% of cows grazing pasture in New Zealand may have subclinical hypocalcemia on the day of calving)