METABOLIC DISEASES

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Out line:

1. CALCIUM RELATED PROBLEMS:
   • PARTURIENT PARESIS (MILK FEVER).
   • BOVINE- NON- PARTURIENT HYPOCALCEMIA.
   • OVINE HYPOCALCEMIC PARESIS.
   • CAPARINE PARTURIENT PARESIS.
   • DOWNER COW SYNDROME.
Out line (cont):

• TRANSIT RECUMBENCY OF RUMINANTS.

• LACTATION TETANY OF MARES (ECLAMPSIA, TRANSIT TETANY).
PARTURIENT PARESIS (MILK FEVER)

- **Etiology:**
  - Hypocalcemia.
Epidemiology:

a) Occurrence:

- Universally
- Dairy cows, rarely beef breeds
- Few cases hrs. before, during parturition
- Most in 48 hrs. afterwards
- Sig # up to 8 days after
- Rare cases up to 8 wks after
- Morbidity 3.58%, recovery in treated uncomplicated 75-85%
b) Risk factors:
- High producing cows (heavily fed before parturition)
- Aged puerperal cows (5-10 years group)
- All breeds, high incidence in some families
- Complete udder emptying during 1st 48 hr. after parturition
- Diets containing
  - Excessive Ca^{++} (> 100g/day)
  - Excessive other cations, Na, K
  - High alkalinity
  - High crude protein
- Late cases: relapses increased by fatigue, excitement, estrus, low feed intake for 24 hrs. diarrhea
c) Importance
  ○ Death losses
  ○ Serious sequels reduce productive life
Clinical signs

a) Prodromal stage:
   - anorexia, milk yield reduced, rumen stasis, scant feces, normal TPR.

b) Excitement stage:
   - Restlessness, hypersensitivity, tremor, tetany, head-shaking, tongue protrude, teeth grinding in some, slight fever, stiff gait, falls easily.
c) **Sternal recumbancy**: Depression, drowsy, sternal recumbancy, unable to rise, limbs flexed normally, lateral neck kink, or head turned into flank, fear gesture when approached (head extended, mouth opened, tongue protruded), hypothermia, heart sound intensity decreased, HR increased to 80/min, pulse small amplitude, difficulty raising jugular vein, breathing normal, muzzle dry, eyes dry and staring, pupil dilated and reflex diminished, ruminal stasis and secondary bloat, constipation and anal relaxation (no reflex).
d) Lateral recumbency: Cow almost comatose, lateral recumbency, limbs stuck out but limbs flaccid, cow unable to sit up, hypothermia, heart inaudible, pulse impalable, HR up to 120/min, animal dies quietly after course of 12-24 hrs.
Complications:

a) Hypomagnesemia: Tetany, hypersensitivity, restlessness persists after early episodes of:
   - Tremor, eyelid twitching, trismus, tetanic convulsions
   - Heart sound loud, heart rate fast, dyspnea
   - Death during convulsions

b) Dystokia: in cows with milk fever at or before calving:
   - Cervix incompletely dilated
   - Uterus atonic
   - Treatment with Ca++ relieves dystokia

c) Uterine prolapse
Clinical pathology:
- Total serum Ca$^{++}$ level below 8 mg/dl (2.0 mmol/1) usually below 5 mg/dl, sometimes below 2 mg/dl
- Mg increased to 4-5 mg/dl (1.62-2.06 mmol/1)
- P decreased to 1.5-3.0 Mg/dl
- CK increased in some

Necropsy:
No significant lesions unless concurrent disease e.g. food aspiration into trachea, lungs.
Diagnosis (DX):
- differentiation from:
  - Downer cow syndrome, Non-parturient hypocalcemia, acetonemia, hypomagnesemic tetany, coliform mastitis, acute diffuse peritonitis, acute septic metritis, aspiration pn, radial paralysis, gastrocnemius muscle rupture, maternal obstetric paralysis, sciatic nerve injury, obturator nerve injury, coxofemoral joint dislocation, Ischemic muscle necrosis.
Treatment (TX):

a) Basic:
   - Ca.borogluconate, 25% solution
     - Small cow 350-500ml
     - Large cow 800-1000ml
     - Half dose IV and half S/C
b) Response:
• positive response include:
  o belching
  o muscle tremor
  o muzzle sweating
  o HR decreased, sounds louder, pulse amplitude increased
  o Defecation of firm, mucus-covered turds
  o Voluminous urination
Response (Cont):

• Incomplete reaction: i.e. improvement in pulse, heartbeat, rumen movement, defecation, muzzle sweat but patient unable to rise due to:
  o Prolonged recumbency before treatment
  o Downer cow syndrome
  o Hypophosphatemia
  o Muscle, nerve, bone conditions listed above under diagnosis.
Response (Cont):

• Adverse reaction: including need to slow or stop injection, reconsider diagnosis:
  o Serious HR increase, serious heart irregularity, sudden death.
  o Causes of adverse reaction include:
    ▪ Too rapid injection
    ▪ Overdosing
    ▪ Presence of toxemia, e.g. coliform mastitis
    ▪ Cow excited, frightened, hyperthermic
Relapse due to:

- Extreme susceptibility of individual cow
- Inadequate dose of Ca$^{++}$
- Complete milking out
c. Supplementary TX:
• Compound parenteral injections including glucose, Mg, P.

• Incomplete milking out for 48-72 hrs.

• Frequent turning from side to side, massage of upper limbs
• Slinging by hips
• Pharmacological, electrical stimulant widely used
Control:

- In low-incidence herds monitor cows closely at parturition time, treat cases early:
  - Consider genetic selection
  - Adjust feeding program
  - Institute a prophylaxis procedure
Control (Cont):

a) **Feeding management:**
   - Use supplement of ammonium sulphate or ammonium chloride, each at 100g/head/day
   - Reduced feed intake in dry period is used but creates risk of acetonemia and pregnancy toxemia
   - $\text{Ca}^{++}$ intake should not be excessive (<100-125 g/head/day) in dry period.

b) **Calcium gel treatment**

c) **Vitamin D analogs**
BOVINE- NON- PARTURIENT HYPOCALCEMIA

- **Epidemiology**:
  - Usually history of grazing green cereal crop.
  - Bouts of diarrhea
  - Rumen stasis due to high grain diet not severe enough to cause carbohydrate engorgement.

- **Signs**
  - Recumbency
  - Recovers with standard Ca$^{++}$ treatment for milk fever.

- **Clinical Pathology**:
  - Serum Ca$^{++}$ levels below 8 mg/dl
OVINE HYPOCALCEMIC PARESIS

- **Etiology:**
  - Depression of ionized Ca$^{++}$ levels in tissue fluids as a result of imbalance between Ca$^{++}$ input from diet plus resorption from bones, relative to Ca$^{++}$ output in deposition in fetuses or secretion in colostrum or milk.
OVINE HYPOCALCEMIC PARESIS (Cont)

❖ Epidemiology:

- As outbreaks in pregnant (last 6 wks) or lactating (first 10 wks) ewes exposed to:
  - Forced exercise
  - Long-distance transport
  - Sudden feed deprivation
  - Grazing on oxalate–rich plants, green cereal crops, low-Ca²⁺ content pasture
  - Outbreaks in dry sheep up to 1 yr old on green oat grazing or short pasture
  - Feedlot sheep on diet supplemented with Mg as a prophylaxis against hypomagnesemia
Clinical findings:
- Early cases stilted, proppy gait
- Tremor of shoulder muscles
- Recumbency in some, not all, sternal recumbency (legs underneath or stretched out behind).
- Head rested on ground
- Rumen flaccid
- Nostrils plugged with dried mucus
- Breathing fast, pulse impalpable
- Terminally flaccid paresis, somnolence
- Death in 6-12 hr without TX
- Dramatic recovery with Ca^{++} TX
Additional syndrome: young sheep may also show poor growth, lameness, bone fragility

Clinical Pathology: total serum Ca++ level <4.6 mg/dl.

Necropsy: no lesions apart from concurrent disease.

Diagnosis (DX):

Can be confused with:
- Pregnancy toxemia
- Carbohydrate engorgement
- Soluble oxalate poisoning
OVINE HYPOCALCEMIC PARESIS
(Cont)

- **Treatment (TX):** 50-100 ml 25% Ca. borogluconate IV or SC.

- **Control:**
  - Avoid high calcium diets in late pregnancy
  - Avoid alimentary tract stasis, feed deprivation, excitement, stress, exposure to inclement weather, transport, sudden changes of feed.
CAPARINE PARTURIENT PARESIS

Similar to bovine parturient paresis in all aspects
DOWNER COW SYNDROME

Etiology:
A complication of hypocalcemic parturient paresis encouraged in its development by:
• A distokia.
• Slippery footing
• Delay in treating patient with milk fever
• Excessive body weight of patient
• Concurrent hypophosphatemia
• Concurrent hypokalemia in creeper cows
• Inadequate dose of Ca++ in TX of milk fever
Downer Cow Syndrome
DOWNER COW SYNDROME (Cont)

Clinical signs:
- Prior history of milk fever in most
- Recumbency, unable to rise for 24 hrs
- Bright, alert; appetite, water intake, defecation and urination normal.
- Most make repeated attempt to rise
- Progress forwards by crawling with forelimbs, hindlimbs drag out behind in frog-like posture, the creepers
- Some patients assume lateral recumbency with opisthotonus but sit normally when raised to sternal recumbency but soon lapse back into lateral position
- Patients still recumbent at 7 days unlikely to rise but some cow can continue for wks if light in weight, carefully nursed.
- Euthanasia usually necessary in laterally recumbent cases.
Downer Cow Syndrome
Complications:

- Acute mastitis
- Decubitus ulcers at major limb joints
- Further injury to limbs
DOWNER COW SYNDROME (Cont)

- **Clinical pathology**
  - No biochemical differences between milk fever cows that become downers and those do not
  - Serum levels of creatinine kinase, ALT, AST, elevated, reducing to normal by day 7.
  - Marked proteinuria (unexplained) in most

- **Necropsy**:
  - Variable degrees of skeletal myopathy in upper hind limbs
  - Variable damage to sciatic, peroneal nerves
  - Unexplained acute, focal myocarditis
Diagnosis (DX):
Other causes of similar syndrome include:
• Radial paralysis
• Gastrocnemius muscle rupture
• Maternal obstetric paralysis
• Sciatic nerve injury
• Coxofemoral joint dislocation
• Ischemic muscle necrosis (degenerative myopathy)
Treatment (TX):
- Parenteral injections of solutions containing Mg, P, Ca
- Corticosteroids
- Vit. E and Se mixture
- Slinging of cows which have some motor functioning remain
- Repetitive electrical stimulation of cattle prod of upper thigh muscles in laterally recumbent patients.
Control:
- Early TX of all recumbent cows, objective of getting them up by 24 hrs
- Frequent turning to other side
- Provide rough, dry footing
- Plenty of bedding
- Early use of sling
TRANSIT RECUMBENCY OF RUMINANTS

• Etiology:
  Physical exhaustion with/without hypocalcemia

• Epidemiology
  a. Occurrence:
  • After prolong transport
    o Late preg. ewes and cows
    o Lambs consigned for feedlots
  • Cows and sheep to abattoirs
  • Morbidity variable, case fatality high
TRANSIT RECUMBENCY OF RUMINANTS

b. Risk factors:
• Severe physical stress
• Prolonged deficiency of feed and water
• Hot weather
• Overcrowding

c. Importance: serious death losses
*Clinical findings:
* Signs while still on train
* Restlessness
* Trismus, teeth grinding, staggering
* Complete anorexia
* Rumen stasis
* Hind limb paresis, paralysis
* Lateral recumbency in lambs
* Reumbency
* Coma, death after 3-4 days course
TRANSIT RECUMBENCY OF RUMINANTS

*Clinical pathology:
*Mild hypocalcemia, hypophosphatemia in cattle
*Hypocalcemia, hypomagnesemia, hypoglycemia in some sheep, many patients have no abnormalities

*Necropsy:
*No significant lesion
*Ischemic myonecrosis in a few

*Diagnosis
*can be confused with:
  *Vitamin E and selenium nutritional deficiency
  *Heat exhaustion
  *Exertional myopathy
TRANSIT RECUMBENCY OF RUMINANTS

• Treatment:
  * Some cases respond to IV fluids containing Ca, Mg salts, phosphates, glucose
  * Large volumes isotonic fluids IV recommended but impractical in the usual circumstances of a group problem, rehydration by oral fluid alimentation effective
  * Corticosteroids as support therapy

• Control:
  * Provide feed and water before and during transport
  * Tranquilizers before travel in nervous animals
  * Minimum feed and water for first 24 hrs after episode
  * Minimum exercise for 3 days
LACTATION TETANY OF MARES (ECLAMPSIA, TRANSIT TETANY)

• **Etiology:**
  * Hypocalcemia

• **Epidemiology:**
  * In lactating mares at about:
    * 10 days after foaling
    * 2 days after weaning
  * Stallions, geldings, dry mares subjected to feed deprivation, physical stress
  * Can be outbreaks with high morbidity, many recover spontaneously but case fatality rate may be 60% especially lactating mares at foal heat or weaning.
LACTATION TETANY OF MARES (ECLAMPSIA, TRANSIT TETANY)

• Clinical findings:
  * Profuse sweating
  * Limb tetany causes unwillingness to move, stiff gait, elevated tail.
  * Dyspnea
  * Wide dilation of nostrils
  * Diaphragm spasm causing thumping hiccup in some
  * Maseter muscle fibrillation, trismus, no 3\textsuperscript{rd} eyelid prolapse
  * Patient anxious, hypersensitive to movement, touch
  * Transient temperature elevation
  * Heart rate fast, irregular
  * Unable to prehend or chew but tries persistently
  * Gut sound reduced
  * No urination, defecation
  * At 24 hrs, recumbency, tetanic convulsions
  * Death after course of 48 hrs

* Prompt, complete recovery after IV Ca\textsuperscript{++} therapy
LACTATION TETANY OF MARES
(ECLAMPSIA, TRANSIT TETANY)

• Clinical pathology:
  * Hypocalcemia (4-6 mg/dl, 1.0-1.5 mmol/l)
  * Serum Mg levels variably increased, decreased

• Necropsy:
  * No specific lesions

• Diagnosis
  * Resembles:
    * Tetanus
    * Colic
    * Laminitis

• Treatment (TX):
  * Slow IV Ca. borogluconate solution to effect

• Control:
  * Avoid transport, physical stress in lactating mares