METABOLIC DISEASES

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Out line:

1. post parturient hemoglobinuria.

2. Paralytic Myoglobinuria (AZOTURIA).

3. Hyperkalemic Periodic Paralysis
post parturient hemoglobinuria

Etiology:
- Hemolytic anemia caused by agents in feed of cattle made susceptible by low P, copper or Se intake.

Epidemiology:
- Occurrence:
  - Adult cows calved 2-4 weeks previously
  - High producing cows in lactations 3-6
  - Morbidity low, usually sporadic, may be outbreak, case fatality 50%.
- Risk factors:
  - Diets deficient in P, or copper or possibly Se.
  - Rape, other cruciferous plants in diet.
  - Lush spring pasture
  - Very cold drinking water
post parturient hemoglobinuria

Clinical findings:
- Anorexia, mild cases may continue to eat
- Weakness, stumbling gait, unwilling to move
- Serious drop in milk yield
- Red black, slightly turbid urine
- Mucosal pallor, possibly jaundice in late stages
- Tachycardia, loud heart sounds
- Exaggerated cardiac impulse, jugular pulse
- Deep respiration, dyspnea in some
- Recumbency, death after 3-5 day course
post parturient hemoglobinuria

• Sequel:
   3 wks convalescence
   Pica
   Gangrene in digits, ear tips in some
   Acetonemia

• Clinical Pathology:
   RBCs, Hb greatly reduced
   Heinz bodies in erythrocytes reported in Cu. deficiency induced disease
   Hemoglobinuria
   Serum P levels often normal, may be as low as 0.4-1.5 mg/dl
   Blood, liver copper levels may be
post parturient hemoglobinuria

• Necropsy findings:
  - Blood thin
  - Carcase jaundiced
  - Hemoglobinuria

• Diagnosis:
  - Similar to other cases of hemoglobinuria
    - Bacillary hemoglobinuria
    - Babesiosis
    - Leptospirosis
    - Rape, kale poisoning
post parturient hemoglobinuria

Treatment:

- Avoid excessive restraint, harassment
- Blood transfusion with minimum of 5 liters
- Sodium acid phosphate solution (60 g in 300 ml water) I/V, same dose S/C, S/C dose repeated 12 hrs for 3 occasions
- Sodium acid phosphate or bone meal subsequently as permanent addition to ration or in lick or salt block.

Control:

- Adequate phosphorus supplementation in diet
- Copper supplementation in copper deficient areas
PARALYTIC MYOGLOBINURIA (AZOTURIA).

Is a disease of horses, occurring during exercise after a period of inactivity on full rations characterized by myoglobinuria and muscle degeneration.

• Etiology:
  Skeletal muscle necrosis caused by lactic acid resulting from anaerobic metabolism of muscle glycogen.
PARALYTIC MYOGLOBINURIA (AZOTURIA).

• Epidemiology:
  - Sporadic cases in performance horses
  - Horses maintained on full grain rations during period (2-14 days) of sudden cessation of exercise, due usually to minor injuries, riding pleasure horses only at weekends
  - After general anesthesia
  - Occasional outbreaks in horses at pasture
  - Sporadic cases in cattle after release from housing in spring
PARALYTIC MYOGLOBINURIA (AZOTURIA).

• Clinical findings:

- Signs commence 15 minutes to one hr after exercise, which need be mild only
- Profuse sweating
- Stiff gait
- Reluctant to move
- Mild cases without hemoglobinuria, if rested completely at this stage, may recover
Clinical findings....

- Lateral recumbency
- Struggling, attempts to rise
- Dyspnea
- Temperature elevated
- Usually both hind limbs involved, may be only one
- Rump, thigh muscles hard, soreness over the area
- Urination suspended
- Urine brown-red
- Death after course of 24 –48 hrs
PARALYTIC MYOGLOBINURIA (AZOTURIA).

Clinical pathology:
- Myoglobinuria
- Casts, protein in urine
- Serum creatinine markedly elevated for several days

Necropsy findings:
- Myonecrosis in gluteal, quadriceps, iliopsoas, vastus muscles causing pale discoloration, with a waxy, cooked appearance
- Dark-brown urine in urinary bladder
PARALYTIC MYOGLOBINURIA (AZOTURIA).

• Diagnosis:
  □ Similar to:
    ✓ Laminitis
    ✓ Nutritional deficiency of Se.
    ✓ Generalized or local myositis
    ✓ Exertional rhabdomyolysis (Tying-up)
    ✓ Iliac thrombosis
Myoglobinuria in horse due to Exertional rhabdomyolysis (Tying-up)
Coffee colored urine collected from a horse with Exertional rhabdomyolysis
PARALYTIC MYOglobinuria (azoturia).

**Treatment:**
- Immediate cessation of all exercise, horse moved by motor transport
- Avoid recumbency, sling if necessary
- Analgesic if pain severe
- Corticosteroids I/V
- Thiamin hydrochloride (0.5 g I/M daily), Se and vit. E preparations, widely used
- Intensive I/V fluid-electrolyte therapy to prevent renal tubular blockage with myoglobin, alkalinization of urine preferred
Control:

Reduce grain ration when horses skip training.

In high-risk situation commence exercise with gentle walking, increased gradually.
HYPERKALEMIC PERIODIC PARALYSIS

Etiology:
- A probable inherited metabolic defect, autosomal dominance of the defect proposed
- Resembles hyperkalemic periodic periodic paralysis in humans

Epidemiology:
- In quarter horses
- 1-5 years old
HYPERKALEMIC PERIODIC PARALYSIS

Clinical findings:

- May be history of bouts of muscle fasciculation or involuntary recumbency
- Intervals between bouts months or years
- Bouts of:
  - Muscle twitching in neck, trunk, spreading to other areas
  - Muscle weakness, swaying, staggery gait
  - Dog-sitting posture
  - Recumbency in some
Clinical findings.....

- Generalized sweating in some
- Some patients apprehensive
- Temperature normal, heart and respiratory rates may be elevated
- Inspiratory stridor in some
- Third eyelid prolapse common
- During attack percussion of muscle with plexor causes sustained muscular contraction
- Spontaneous recovery after course of 30-60 minutes, rarely some hours.
HYPERKALEMIC PERIODIC PARALYSIS

• Clinical pathology:
  - Serum $K^+$ levels elevated to 5.0 – 11.7 mEq/liter
  - PCV, total plasma protein concentration increased
  - Electrographic abnormalities in clinically normal patients is sensitive test for the disease

• Diagnosis:
  - Similar to:
    - Exertional rhabdomyolysis
    - Abdominal colic
    - Esophageal obstruction
    - Upper respiratory tract abnormalities
HYPERKALEMIC PERIODIC PARALYSIS

Treatment:

- Recovery spontaneous
- Recommended treatment during attack include:
  - Na. bicarbonate 2% solution I/V at 1mEq/kg or
  - Dextrose solution 5% (4.4-6.6 ml/kg) or
  - Ca. borogluconate solution (23% at 0.2-0.4 ml/kg) diluted in 1-2 liters of 5% dextrose
Control:

- Acetazolamide (2.2mg/kg) orally every 8-12 hrs controls subsequent episodes
- Feed oaten instead of alfalfa hay, grain 2-3 times daily free access to salt.
- Cull affected animals identified by electromyography.
LOW MILK FAT SYNDROME:

- Milk fat often reduced to less than 50% normal, in normal milk yield
- Caused by low fiber diets, lush pasture, finely ground grain, as meal or pelleted.