B- ENERGY RELATED PROBLEMS

1. KETOSIS OF RUMINANTS

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ACETONEMIA OF CATTLE:

• *Etiology*:
  Negative nutritional balance caused by inadequate nutrition
• *Epidemiology*:

**Occurrence**

- Disease of dairy cattle
- Commonest in housed cows in winter
- 90% of cases in 1\textsuperscript{st} 60 days, most in 1\textsuperscript{st} 30 days, of lactation
- Cows of any age, rare at 1\textsuperscript{st} lactation, peak at 4\textsuperscript{th}.
- Morbidity low, case fatality nil.
Epidemiology:

Risk factors:
- Low energy intake
- High protein intake
- Excessively fat cows
- Intetercurrent disease limiting feed intake e.g. milk fever, foot rot.

Importance
- Production loss during illness, poor return to full lactation in untreated cases.
• **Clinical findings:**

A. **Wasting syndrome:**

- Gradual, moderate decreased milk yield, appetite during 2-4 days.
- Grain not eaten, hay is
- Pica in some
- Weight loss
- Cutaneous elasticity lost
Clinical findings
A. Wasting syndrome ......

✓ Feces firm, dry
✓ Moderate depression, disinclined to move
✓ T.P.R normal
✓ Ruminal movement decreased
✓ Ketone odor on breath; in milk
✓ May be transient bouts staggering, blindness
✓ Spontaneous recovery, but milk yield not regained
B. Nervous syndrome:
• Depraved appetite
• Chewing movement with salivation
• Hyperesthesia, bellows on stroking, pinching
• Moderate tremor, tetany in some
• Gait incoordinated
• Self injury in some
• Bouts recur at 8-12 hrs intervals
B. Nervous syndrome.....

- Depraved appetite
- Chewing movement with salivation
- Hyperesthesia, bellows on stroking, pinching
- Moderate tremor, tetany in some
- Gait incoordinated
- Self injury in some
- Bouts recur at 8-12 hrs intervals
C. Subclinical disease:

- Ketonuria
- Milk yield depressed
- Reduced fertility, ovarian dysfunction, endometritis.
Clinical pathology:

✓ Hypoglycemia- (20-40 mg/dl), severity of clinical signs closely related to severity of hypoglycemia

✓ Ketonuria 80-1300 mg/dl, basis for field test using tablets, paper strips, positive test purple color in 30 seconds; primary ketosis cases give deep purple, 2ndry cases give mauve color.

✓ Ketonemia 10-100 mg/dl
**Clinical pathology**

- Milk ketone levels increased up to 40 mg/dl
- Liver glycogen levels low, glucose tolerance curve normal
- Blood volatile fatty acid levels very high
- Ruminal butyric acid elevated relative to acetic, propionic acids
- Mild hypocalcemia 9 mg/dl
• **Necropsy findings:**
  - Fatty degeneration of liver

**Diagnosis**

**A. Wasting syndrome may be confused with:**
  - Traumatic reticuloperitonitis
  - Bovine pyelonephritis
  - Indigestion
  - LDA

**B. Nervous syndrome may be confused with:**
  - Listeriosis
  - Rabies
  - Sporadic bovine encephalopathy
  - Hepatic encephalopathy
  - Polioencephalomalacia
Treatment

A. Replacement therapy:
✓ I/V injection 500 ml 50% glucose solution, rapid improvements, relapse common.
✓ S/C injection concentrated glucose not recommended
✓ Plus oral propylene glycol or glycerin (225 g twice daily for 2 days, then 110 g daily for 2 days), by drench or in feed but some cows refuse to eat it.
✓ Alternative to propylene glycol, Na. propionate, lactates also effective but response slow.
Treatment

B. Hormonal therapy:
- Glucocorticoids, e.g. dexamethazone, produce hyperglycemic effect in 24 hrs, lasting 4-6 days
- Insulin used in cases unresponsive to corticosteroid, glucose therapy
Control

✓ Cows should be in moderate condition at calving (<10% with body condition score 4)
✓ Best preventive feed in late pregnant cows is ground maize
✓ Housed cows should get some exercise every day
✓ Adequate cobalt, copper, P, iodine in ration
✓ Prophylactic feeding Na. propionate (110 g/day), propylene glycol (150 ml/day), glycerin, from calving for 6-8 weeks.
Prophylactic feeding Na. propionate (110 g/day). propylene glycol (150 ml/day), glycerin, from calving for 6-8 weeks.
Control....

✓ Monensin has prophylactic effects but needs care to avoid side effects
✓ Monitoring of blood glucose or beta-hydroxybutyrate, or urine ketones and improvement in herd diet, or individual cow treatments instituted when indicated may be profitable in problem herds.
EWE PREGNANCY TOXEMIA

Etiology:
Negative nutritional balance due to inadequate nutrition in late pregnancy in twin or large single pregnancy ewes
Epidemiology

Occurrence:

- Widespread in all sheep industry, especially intensive system.
- Morbidity very variable, outbreaks due usually to management error, e.g. penning ewes without feed for a day, changing to different kind of feed.
- Case fatality rate with or without TX close to 100%.
Epidemiology....

Risk factors:

- Late pregnant ewes, last month
- Adult ewes carrying multiple lambs or large singles
- Decline in plane of nutrition during last 2 months of pregnancy in previously well-fed ewes, sudden further deprivation during management procedures e.g. crutching transport, often precipitates severe outbreak 48 hrs later
- Overfat ewes
- Intercurrent disease, e.g. foot rot, reducing feed intake

Importance:
Severe death losses, usually in studs or intensively managed farms
triplets pregnant ewe
• **Clinical findings:**

A. Early stages:

- Separation from flock, alert bearing, disinclination to move (all indicating blindness)
- Walks into objects, head-presses
- May lab water continuously
- Constipation
- Teeth-grinding.
Clinical findings:
Early stages …..

- Brief convulsive episodes, often missed
  - Lip- twitching
  - Jaw-champing with salivation
  - Cog-wheel dorsiflexion, or lateral deviation of head
  - Circling in either direction
  - Recumbency, tonic- clonic convulsion
Recumbent ewe due to pregnancy toxemia
B. Late stages:

- Somnolence
- More visible convulsive episodes
- Star-gazing posture
- Incoordination, easy falling
- Ketone smell on breath
- Recumbency
- Fetal death common, followed by toxemia in ewe
- Death in coma after course of 6-8 days
• **Clinical pathology:**
  - Hypoglycemia early, terminally blood glucose levels normal to high
  - Ketonemia, ketonuria
  - Severe metabolic acidosis
  - Terminal uremia
  - Dehydration
  - Elevated plasma cortisol levels (>10 ng/ml terminally)
Necropsy findings
- Severe fatty degeneration of liver
- Twin lambs or large single, usually dead
- Poorly defined renal lesion
• **Diagnosis**
  - Needs to be differentiated from:
    - Hypocalcemic paresis
    - Listeriosis
    - Rabies
    - Brain abscess
    - Coenurosis
    - Otitis media
    - Louping–ill.
• **Treatment:**
  - Response to TX.s recommended for bovine acetonemia unsatisfactory
  - In ewes still able to stand 25 mg dexamethazone effective, may be due to birth induction resulting
  - Good results recorded with anabolic steroids, e.g. 30 mg trenbolone acetate
  - Other expensive complicated Tx.s reported
  - Removal of fetus by hormonal induction, caesarean but only in early stages before condition irreversible
• **Control:**

- Avoid management errors, especially ensure nutrition plane rising, no forced inanition, ensure weight gain of 10% in ewes carrying single lambs, 18% for those with twins, during last 2 months of pregnancy
- Examine flock daily, treat suspects with propylene glycol orally, start supplementary feeding
KETOSIS IN GOATS
• Both acetonemia type (as in cows) and pregnancy toxemia type (as in ewes) syndromes occur.
• Exaggerated dominance, submissive attitudes in goats may lead to inadequate feed intake by some does where herd fed heavily on grain in self–feeders.
FATTY INFILTRATION OF THE LIVER IN CATTLE (FAT COW SYNDROME, PREGNANCY TOXEMIA)
• **Etiology:**

  – Mobilization of excessive quantities of fat from body depots and their deposition in the liver because of:
    
    • Deprivation of feed in fat, heavily pregnant beef cows or
    
    • sudden demand for energy in early lactation dairy cows
Epidemiology:

• Occurrence:
  • Dairy of beef herds
  • Increased occurrence in farms with erratic or high risk management
  • Morbidity about 1%, case fatality rate about 100%
**Epidemiology**

– **Risk factors:**

• A disease caused in dairy cows by the management practices aimed at maximizing milk yield per cow, e.g. challenge feeding in which feed intake is maximized during late pregnancy and increments of feed are added to the diet as milk yield increases, some very fat cows result.

• Young cattle most affected

• Cows carrying twins

• Abnormally long dry periods

• Feeding in groups

• Sudden reduction of feed in last 6 weeks of pregnancy to reduce dystokia rate, especially in cows with twins
Clinical findings:

• Puerperal dairy cows:
  – Usually a history of intercurrent diseases e.g. retained placenta, LDA
  – Incomplete recovery from primary disease
  – Complete anorexia
  – Staring gaze, opisthotonus, tremor in some
  – Severe ketosis
  – Terminal tachycardia, coma
  – Death in 7-10 days
Clinical findings....

• Late pregnant beef cows:
  – Aggression
  – Restlessness, excitement
  – Stumbling gait, fall easily
  – Terminally recumbency
  – Scant firm feces, yellow diarrhea terminally
  – Tachycardia
  – Anorexia
  – Depression
  – Respiration rapid, expiratory grunt
  – Clear, profuse nasal discharge
  – Flaking muzzle skin
  – Cows die quietly after 10-14 days
Marked fatty infiltration of the liver
• **Clinical pathology:**
  - Hypoglycemia, ketonemia, ketonuria
  - Serum non-esterified fatty acids increased
  - Increased blood levels of betahydroxybutyrate
  - Elevated serum bilirubin, liver enzyme levels
  - Liver fat levels in biopsy specimens assessed by estimates of triglyceride content.
• **Necropsy findings:**
  – Liver grossly enlarged, pale yellow, friable, greasy
  – May be intercurrent disease, usual cause of death in fatal cases
• **Diagnosis:**
  – Lactating dairy cows differentiate from:
    • LDA
    • Downer cow
    • Acetonemia, wasting syndrome
    • Parturition syndrome
  – Late pregnant beef cows, disease resembles:
    • Abomasal impaction
    • Vagus indigestion
    • Chronic peritonitis
• Treatment
  – Prognosis guarded, especially for anorexic cows
  – Intensive intravenous fluid, electrolyte therapy or water plus balanced electrolytes intraruminally
  – Intraruminal infusion of 5-10 liters rumen juice from normal cows
  – Corticosteroids e.g. dexamethazone 20mg, every second day, or anabolinic steroids
• **Control:**
  
  – Avoid overfatness during dry period or 3rd trimester of pregnancy, by sorting cows into groups on basis of body score, feeding appropriately.

  – Treat puerperal diseases early
EQUINE HYPERLIPEMIA

• Etiology:
  – Deficient metabolizable energy in pony mares during late pregnancy, early lactation
Epidemiology:

- Occurrence:
  - Pony mares, rarely in geldings, stallions secondary to other disease
  - All ages sporadic, some outbreaks
  - Most cases occur 4-8 weeks after foaling
  - Case fatality rate 65%
Epidemiology ....

- **Risk factors:**
  - Recent transport
  - Concurrent sand colic parasitism
  - Falling nutritional plane during late pregnancy, early lactation
• **Clinical findings:**
  - Depression, anorexia, weight loss
  - Muscle fasciculation (limbs, trunk, neck)
  - Ventral edema in some
  - Compulsive walking, mania in some
  - Continuous lapping or attempts to drink but unable to swallow
  - Heart and respiratory rate increased
  - Persistent diarrhea terminally
  - Thick, porridgy, fetid feces
  - Somnolence, coma
  - Abortion may precede recovery
  - Death after course of 6-8 days
• Clinical pathology:
  – Milk – like opalescence of serum, plasma
  – Total lipids greatly increased up to 4-8 g/dl of serum
  – Blood glucose normal
  – Leukocytosis, neutrophilia
  – Renal, hepatic function tests abnormal
  – Irreversible metabolic acidosis in late stages
Hyperlipemia (plasma, compared to normal horse on left)
• *Necropsy findings:*
  – Fatty change in most internal organs
  – Enlarged, yellow-orange liver
  – Widespread vascular thrombosis, generalized skeletal muscle degeneration
166 Hyperlipemia (liver, cut surface).

**Note:** Pale, waxy appearance. Liver grossly enlarged with rounded edges and a piece floated in 10% formol saline.
• **Diagnosis:**
  – May be confused with:
    • Mare hypocalcemia
    • Congestive heart failure
    • Hepatitis
• Treatment:
  – Identification, treatment of primary disease
  – Parenteral administration of glucose valuable in very early stages
  – Insulin 30 iu parenterally with 100 g glucose orally then, 15 iu insulin plus 100 g glucose on alternate days
  – Correction of acidosis
• **Control:**
  – Adequate control program for nutrition, parasite near parturition
  – Avoid transport of ponies during this period
  – Periodic blood sample examination for hyperlipemia