Bovine neurological disease

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WCVM
Bovine neuro cases are hard.......  

• Often lacking specific history
• Classic neurological exam not really possible
• Limited diagnostic tests readily available
• There are some important disease you don’t want to miss
Presenting signs

• Blindness
• Recumbency
• Circling/head tilt
• (change in behaviour)
Neurological examination

• Mental status
• Cranial nerves
• Gait and posture
• Spinal reflexes
• Pain perception
Supportive diagnostic tests

• CBC
• Chemistry
• CSF
• Lead
Another useful diagnostic tool

• Time

• Rate of disease progression and response to treatment can be very helpful

• E.g.
• Rabies rapid progression to death (less than 3-5 days)
• BSE very, very slow progression (months)
2 common identifiers

• Blindness

• Circling/head tilt
Blindness

- Lead
- Polio
- Vitamin A
- Pituitary abscess
Lead

• Mainly batteries and paint
  • Also lubricants, roofing, lead shot

• Studies indicate 4-12% of the herd may be affected but asymptomatic despite levels >0.35ppm (safe consumption 0.05ppm)
Lead poisoning in Cattle

• Common

• Signalment
  • Any animal, any age (calves may be slightly more common)
  • Any bizarre behaviour patterns
Lead Poisoning
Distant Examination

- Ataxia (drunkenness)
- Weakness (recumbency)
- Convulsions
- Aimless wandering
- Headpressing
- Opisthotonus
- Teeth grinding
- Bellowing
- Hyperasthesia
Sources of lead

• Car batteries
Sources of lead

• Lead paint
  • White and green
Other sources of lead

• Crank case oil

• In all cases the owner will deny that there is any possible that the animals could have been exposed to lead
General Physical Examination

- Disruption of normal GI function
  - Decreased rumination
  - Mild bloat
  - Tenesmus
  - Constipation or diarrhea

- Blindness
Pathophysiology

• Acute vs Chronic exposure

• Acute is much more common
  • 400-600mg/kg in calves (2oz)
  • 600-800mg/kg in adults (1/2 lb)
Confirming Diagnosis

• Blood lead levels
  • Must use heparinized blood sample
  • Blood levels can vary widely in 24 hours
    • Normal 0.05-0.25ppm (death at about 1ppm)

• High powered radiographs of the reticulum may indicate elemental lead
Confirming Diagnosis

• Post-mortem examination
  • Lead in reticulum and rumen

• Check lead levels in;
  • Kidney
  • Liver
To treat or not to treat

1. Remove access to lead
2. Chelation therapy
   1. Ca-EDTA to promote excretion
3. Thiamine
4. Rumenotomy
Problems of the treated cow

• When is it safe to eat?
Polioencephalomalacia

- Aka:
  - PEM
  - Polio
  - Cerebrocorticalnecrosis
    - CCN

- This disease should really be split in two
  - Thiamine deficiency
  - Sulfate toxicity
Polio
Distant examination

• Blindness
• Ataxia (drunkenness)
• Tremors
• Recumbency
• Convulsions
• Opisthotonus (star gazing)
Polio - Pathophysiology

- Thiamine is normally made in rumen
- Needed for pentose phosphate pathway of glycolysis
  - Main energy producing pathway in the brain
Thiamine deficiency

• Thiamine antagonists
  • Amprolium – used for coccidiosis

• Rumen thiaminases
  • Produced by bacterial fermentation
    • *Bacillus thiamainolyticus*
    • *Clostridium sporogenes*
  • Ingested pre-formed
    • Bracken fern
Diagnosing Polio

• Signs are essentially similar to lead poisoning

• GI motility is normally OK

• Pupillary light reflex is maintained
Blindness in polio

• Pupillary light reflex
  • Simple reflex in the brain stem

• Menace response
  • Learned response using higher brain function
Finalizing the diagnosis

- No simple lab tests
  - Erythrocyte transketolase

- PM examination of the brain
  - Cerebral edema
  - Mild yellow discoloration

- Fluoresces under UV light
Treatment of Polio

- Give thiamine (lots of it)!

- 10mg/kg IV then 10mg/kg IM every 3 hours for 5 treatments

- Prognosis good if caught early
Sulfate toxicity

Syndrome cannot be distinguished clinically or at PM from thiamine deficiency
Sources of sulfate

- Water
  - Slough water may be particularly bad especially in drought years
  - Easily tested
  - The exact cut off is not clear
    - 2000-7000ppm is the region
Sources of sulfate

• Feed
  • Hay and grain grown in high sulfate areas will contain more sulfate
  • Certain plants store sulfate
    • Kochia
    • Canadian thistle
Sources of sulfate

• Just when you think you have it straight

• Copper and Molybdenum interfere with sulfate absorption and may be protective
How sulfate is toxic

Not entirely clear

Best theory

- Sulfate is converted to H$_2$S in the rumen. This is eructated and inhaled, absorbed by the lung and transported to the brain in the blood, where it exerts a toxic effect
Sulfate toxicity

• No definitive clinical diagnosis

• Thiamine treatment does not work
Vitamin A deficiency

• Actually more than one syndrome to consider
  • Adult cattle on scrub pastures
  • Feedlot steers
  • Neonatal calves
Presenting signs

• Night blindness – rarely recognized
• Blindness – esp feedlot steers
• Weak animals dying shortly after birth
• Reproductive problems
• Poor hair coat
• Paralysis
• Convulsions
History and epidemiology

• Vitamin A is easily produced from β-carotene
• Carotenes are freely available in any green coloured feed
Syndromes recognized

• Neonates
  • Vitamin A crosses the placenta poorly
  • Calves are heavily dependent on colostrum

• Pastured animals
  • Takes months for adults to develop signs due to good liver stores
 Syndromes recognized

- Feedlot animals 6-12 months age

- Toxicity of chlorinated napthalenes
  - Block production of vitamin A
Pathophysiology of Vitamin A deficiency

• Vitamin A required for:
  • Maintenance of epithelial surfaces
    • loss of secretory capacity
    • replacement with a keratinized epithelium esp respiratory and UGT, cornea (xerophthalimia)
  • Thickened arachnoid results in decreased CSF absorption
  • Production of retinol – night blindness
Neurological signs

• Increased CSF pressure due to:
  • Thickened arachnoid
  • Failure to remodel bone as the animal grows
    • Also puts pressure on cranial nerves esp II
Diagnosis

• Blindness
  • Careful optho exam should reveal papilledema
• Convulsions and paralysis
• Measure CSF pressures with a direct manometer
• Response to therapy
• Measure plasma Vitamin A levels
Diagnosis (2)

• Liver biopsy

• Diet calculations
  • Look at the diet for green coloured feeds
  • Signs typically seen with grains, straw, poor quality hay
Treatment

• Injectable Vitamin A

• Prognosis is good for calves with improvement with 48 hours
Pituitary Abscess

• Bacteria can easily localize in the region of the pituitary

• Open mouth
• Protruding tongue
• Blindness
• Decreased milk production
Pituitary abscess

• Treatment
  • Prolonged antibiotics

• Prognosis
  • poor
Circling

• Listeria

• Middle ear disease
Listeria

• Classic presentation
  • Head tilt
  • Circling
  • Unilateral Facial Paralysis
Listeria

- *Listeria monocytogenes*
- Saphrophic
- Mainly found in poor quality silage
Listeria

• Bacteria enters at gingiva
• ascends to the brain stem through CN-V
• Micro-abscess formation
  • CN VII and VIII

• Treatment
  • Prolonged antibiotics
    • Penicillin
    • Oxytetracycline
Middle ear disease

- Calf dz. assoc. With weaning
- *Histophilus, mannheimia* and *mycoplasma* (esp. dairy calves)

- Dairy calves 2-8 wks age
- More common in the winter

- True incidence unknown as mild cases are often missed
• Middle ear disease can occur in catastrophic outbreaks with morbidity up to 80-100%

• Associated with cross contamination in bottle raised animals

• Associated with URT infections

• Colonization of the pharynx ascending the eustachian tube
  • Rupture of the tympanic membrane
  • Infection of inner ear and osteomyelitis and meningitis
Clinical signs

• Facial nerve paralysis and head tilt/circling

• Purulent aural discharge in many cases
• Head rubbing, head shaking – ear trauma

• Confirm diagnosis with a deep ear swab
  • Otoscope, imaging
Treatment

• Antibiotic therapy
  • Limited evidence – best choices appear to be tulathramycin and enrofloxacin for at least 2 weeks

• Prognosis
  • Better in young calves with early treatment
  • May have persistent signs after treatment
Rabies

• Clinical signs
  • Salivation
  • Bellowing
  • Aggressiveness
  • Paresis/paralysis
Epidemiology

• Vector
  • Skunk
  • Raccoon
• Incubation period
  • 2 weeks – months
• Clinical course
  • 5 days
What to do if you suspect rabies

• Reportable disease
  • Contact Manitoba Ag
  • Contact public health if human exposure
• Isolate and observe animal
• After death or euthanasia
  • Get brain and give to Manitoba Ag.
BSE

• Signs
  • Nervousness
  • Aggression
  • Ataxia

• Slowly progressive to recumbency and death

• Much easier to diagnose in a dairy cow!
What does BSE look like?
Tetanus

• A disease of antiquity that is coming back!

• Many multivalent clostridial vaccines no longer contain tetanus
  • Combined with the use of banding for castration this a bad combination

• Signs
  • Muscle rigidity – often most noticeable in the hind limbs
    • Typically history of surgery, multiple animals affected, lack of vaccination Hx

• Treatment
  • none
Conclusion

• Neurological disease in the bovine is challenging
• There are only a few “real” differentials of consequence
  • Separating them out is not impossible
  • Using clinical signs
  • Diagnostic tests
  • Time
  • Response to treatment
Questions