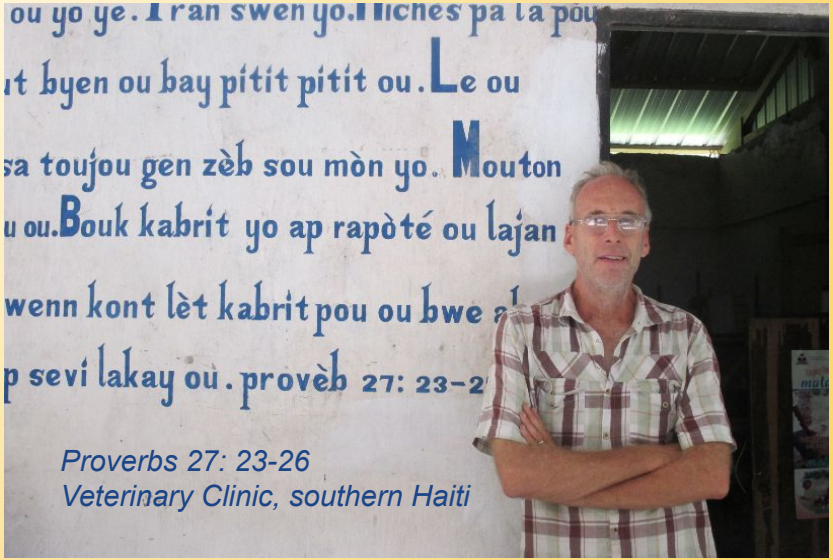
Neurological Diseases in Goats

Paul G Rudenberg DVM

Neurological Diseases of Goats

This lecture will present an extensive differential based on certain neurologic signs in goats, and then focus on several of the common diseases and problems that present these signs.

The author has limited experience on the Indian subcontinent, so some aspects of this presentation may focus on problems found more commonly in the US or the Caribbean.



Proverbs 27: 23-26
Veterinary Clinic, southern Haiti

Differential diagnosis based on common signs

Circling:

Cerebral or vestibular disease:

Listeriosis

Polioencephalomalacia

Coenurosis

Brain abscess, encephalomeningitis (bacterial)

Rabies

Otitis media, vestibular disease

CAE

Cerebral nematodiasis - Setaria digitata

Sinusitis - aberrant nasal bot migration, etc

Head trauma, Cerebral hematomas

Toxic plants, other toxins- nitrofuran, chlorinated hydroccarbons

Differential diagnosis based on common signs

Convulsions:

Infectious - Enterotoxemia (Clostridium C and D)
Pseudorabies, borna, tetanus,
bacterial meningoencephalitis,

Parasitic - Coenurosis (gid), aberrant bot migration,

Metabolic - Polioencephalomalacia, hypomagnesemia, pregnancy toxemia, hypoglycemia, hepatic encephalopathy,

Toxic - toxic plants, overdose with lidocaine, Urea overdose insecticides/pesticides, heartwater (africa) levamisole, herbicides, wood preservatives, Lead Poisoning (rare in goats) and salt poisoning

Differential diagnosis based on common signs:

Ataxia/ incoordination. Numerous causes!

Infections: Listeriosis, , brain abscess/ meningoencephalitis,
Caprine arthritis/encephalitis (low prevalence in India), rabies, scrapie, spinal abcess

Parasites: Coenurosis, Setaria digitata (filarial worm), nasal bot migration, tick paralysis

Metabolic: Enzootic ataxia (copper deficiency)
Polioencephalomalacia, hypomagnesemia,

Toxic chemicals, and toxic plants, lymphosarcoma,

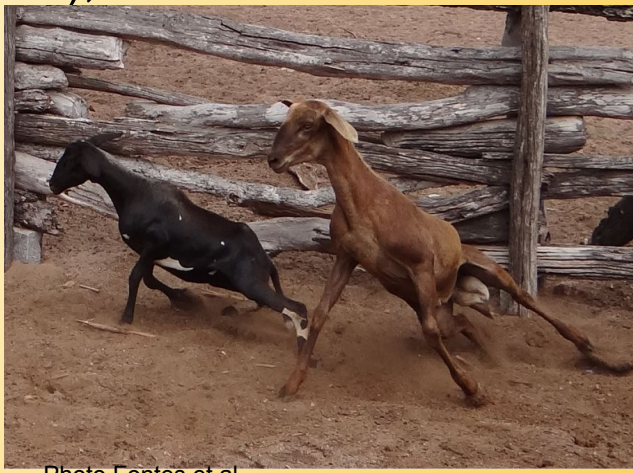


Photo Fontes et al
https://doi.org/10.1590/1678-5150-PVB-6298

Bakarwal goats and sheep, near Srinagar 2013 on journey to Kargil, Ladakh

Listeriosis

Bacterium *Listeria monocytogenes*.- found in soil, water, plant litter, silage and digestive tracts. Zoonotic disease

Signs:

Encephalitic form most common in goats. Unilateral neurologic signs, esp. associated with cranial nerves.

Usually goats are at least 6 months in age.

Circling, leaning or stumbling, head pulled to flank with rigid neck, unilaterial facial paralysis,
ear droop, slack jaw, salivation.

Varies depending on location of microabcesses.

Depression, decreased appetite -dehydration, fever
Abortions may also occur.

Septicemic form also exists. Meningitis may be present

Listeriosis

Transmission Feeding poorly fermented (higher pH) or spoiled silage,etc. In other cases, the source of infection not found.

Enters cranial nerves through wounds in buccal cavity, then to brainstem

Stresses of parasitism, dramatic weather changes and/or advanced stages of pregnancy.

Treatment Must treat 10-14 days!

Penicillin Proc G (40,000 IU/kg BID) IM to - high dose needed cross blood-brain barrier, and because of intracellular nature of *L monocytogenes.*

or IV oxytetracyline 5-10 mg/kg BID sometimes used.

or Florfenicol 20 mg/kg IM every 2 days.

Anti-inflammatories, supportive therapy (fluids and electrolytes) and supplemental feeding.

Listeriosis

Prevention/ Control Discard discoloried, spoiled silage.

Organism can survive in feces, silage, feed, for 5 years, but easily killed with disinfectants. Floors and pens should be thoroughly cleaned and disinfected.

Isolate aborting does from the herd and raising kids separately.

Goat milk and goat products may be human source of Infection - unpasteurized goat milk if does infected, poorly cooked meat

Handling aborted fetuses, placentas and reproductive discharges - gloves and face masks as well as careful disposal.

Polioencephalomalacia

Caused by a **thiamine (vit B1) deficiency** Thiamine, produced by rumen bacteria, needed by brain to metabolize glucose to maintain neuronal osmotic gradients.

Commonly 2-6 month old kids on high concentrate diet.

But can occur at any age.

dairyfarminghut.com

Excessive dietary **sulfate/** high sulfur water can cause similar disease

Related to feeding that disrupts rumen microbes (that produce B1), such as chronic or acute acidosis or indigestion, excess concentrate, moldy hay, weaning stress, molasses in feed, possibly bracken fern (Pteridium).

(In North America, seen especially in winter- increased grain feeding to meet increased energy needs.)

.

Polioencephalomalacia

Signs of illness are related to damage, inflammation and necrosis of cerebrocortical gray matter

Blindness, head pressing, opisthotonos

Incoordination/ataxia,

Rapid, involuntary nystagmus, dorsomedial strabismus,

At onset - diarrhea, anorexia.

Muscle rigidity/tremors convulsions,

Recumbency, coma,

if untreated, the animal generally dies within 24-72 hours. ,

Usually on a few animals in herd, though sulfate excess can be higher percentage.

Polioencephalomalacia

Treatment: **Thiamine** is the only effective therapy. Multiple doses (10 mg/kg body weight three to four times day) of thiamine. First dose IV, followed by IM doses. (Don't use "B-complex" vitamins - not enough thiamine- B1).

Reduce doses as goat improves and regains appetite. Treatment can result in improvement within 2- 24 hours if caught early.

If cerebral edema suspected, give furosemide, mannitol, or dexamethasone(0.1mg/kg). Diazepam to control seizures.

If not responding in 3 days, euthanize.

Control: Control measures include feeding as much roughage and as little concentrate as needed to meet nutritional requirements performance goals.

Avoid excess high-carbohydrate feeds. Prevent accidental access to unlimited amounts of carbohydrates, such as grain bins, feed sacks or tree fruit drops. Avoid moldy feeds. In high-risk herds where animals are on high grain diets, supplement with thiamine mononitrate, probiotics or brewer's yeast

Caprine Arthritis Encephalitis Virus (CAE)

5 major forms of CAE in goats:

polysynovitis/ arthritis - most common in adult goats

encephalitis (inflammation of the brain) - most common in kids

interstitial pneumonia,

mastitis (hard udder),

and chronic wasting- can occur either separately or in addition to any other form of CAE.

Transmission: Spread from mother to kid through the ingestion of colostrum or milk.

Also spread among adult goats through contact with body secretions including blood and feces

Decreases the lifetime productivity, and a barrier to exportation of goats

Caprine Arthritis Encephalitis Virus (CAE)

Encephalitic CAE - *leukoencephalomyelitis*

*(most common form in **kids** starting at 2-4 months) Incoordination*

- *Progressive paresis, paralysis,*
- *Proprioceptive deficit*
- *Depression*
- *Blindness*
- *Head tilt*
- *Proprioceptive deficits*
- *Seizures, Death*

Arthritic CAE

(most common form in adults)

- *Lameness (may be sudden)*
- *Stiffness, Swollen joints,*
- *Reluctance to walk, walking on knees*
- *Abnormal posture*
- *Reluctance to rise*
- *Weight loss*

Caprine Arthritis Encephalitis Virus (CAE)

Most CAEV infections are subclinical.

Diagnosis - Clinical signs, when present, and
Testing - indirect serology- ELISA and Agar gel immunodiffusion tests.

Antibodies remain for life. False positives and negatives exist!

Rare among indigenous goat breeds in lower/middle-income countries, except around imported goats. 3.3% of goats in India in one study (2015 Waseem, Pawaiya, Singh, Gupta). Widespread (seroprevalence rate > 65%) among dairy goats in most high-income economies

Prevention : No vaccine exists.

No treatment exists - cull! Repeated testing, and culling, don't purchase from untested farms.

Prevent of nursing by infected does (keep frozen colostrum available), Avoid feeding pooled milk.

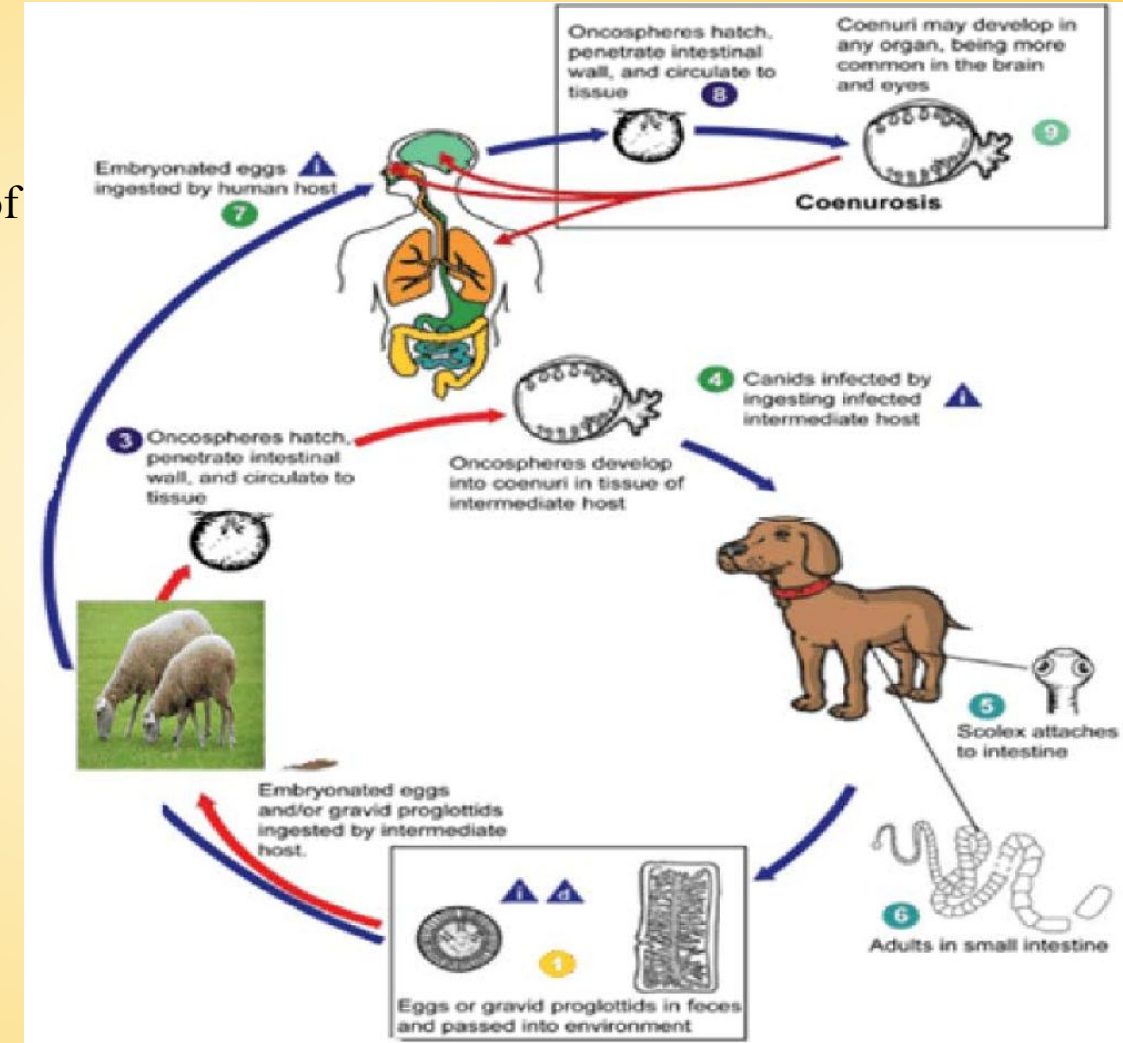
Annual Bakarwal migration, Sind Valley, North of Srinagar

Coenurosis (gid or sturdy)

Zoonotic disease caused by migration of Coenurus cerebralis - Larval stage of Tapeworm - **Taenia multiceps**

Lives in the small intestines of carnivores as definitive hosts. Intermediate hosts are infected via ingestion of contaminated grass by spread eggs from the carnivores feces Leads to cyst formation in different organs [1].

Chart from Review on Cerebral Coenurosis in Small Ruminants Misretaw Gashe, M. Sewalem



Coenurosis (gid or sturdy)

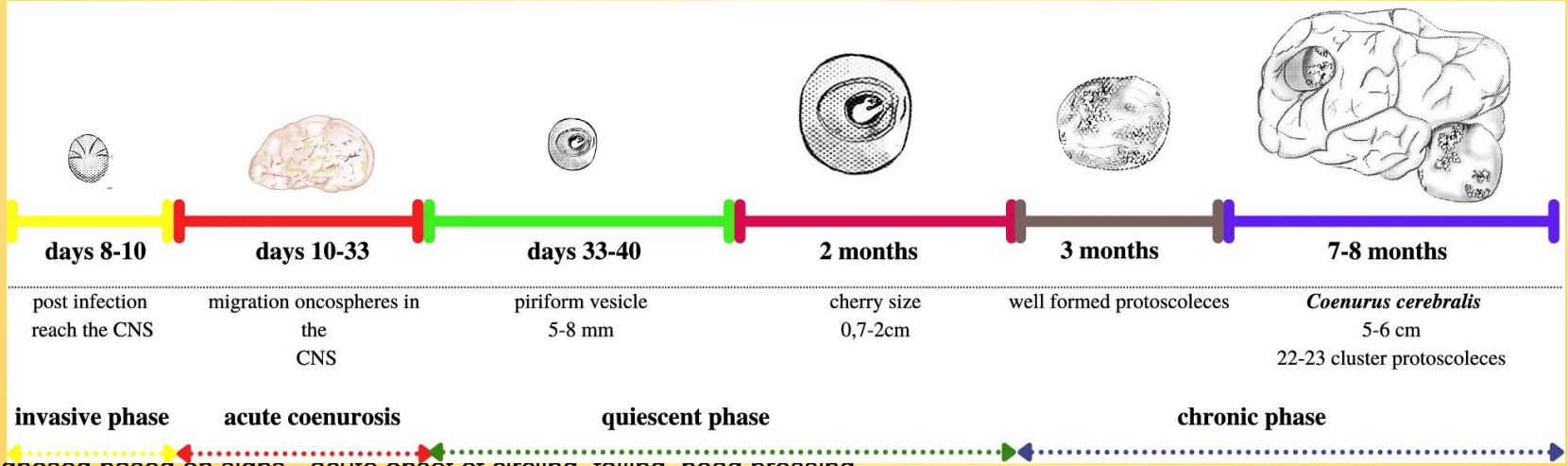
Common in small ruminants, but rare in horses and cattle .

Usually in the cerebrum in sheep but extracerebral tissues in goats

However cysts in the brain of goats and other tissues apart from the brain of sheep have been confirmed, recently.

Non-cerebral coenurosis parasite is also called *Coenurus gaigeri* in goats

Development in the CNS:



Diagnosed based on signs - acute onset of circling, falling, head pressing

Coenurosis

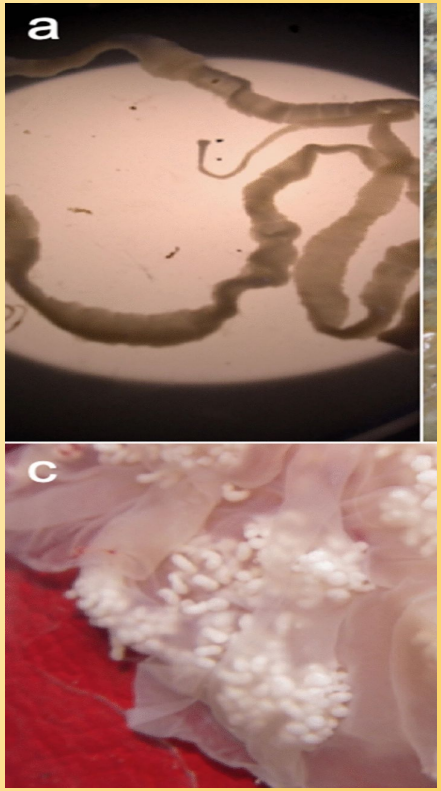
Migration of this parasite causes high economic losses in the small ruminant production, breeding.

Severe infection leads to the death, and discard of organs/ meat.

Zoonotic disease - humans act as incidental intermediate host and may be infected by ingestion of eggs in result of poor personal hygiene

B. Sarma and J. Das -technique of surgical extraction of coenurus cyst from the brain under local anaesthesia -no postoperative complications (!)

Prevention - Reduce stray dogs, treat dogs kept with livestock for tapeworms regularly. Keep goat offal away from dogs.



Photos From: *Taenia multiceps* coenurosis: a review

Meningoencephalitis / brain abcesses

Young kids - from naval infection, esp E coli - may see many other than neuro signs - umbilical infection, arthritis, pneumonia, shock

Contributing factors - lack of colostrum, untreated navel, poor sanitation, bedding, weather stress, poor nutrition and from heat damage due to dehorning.

Adult goats - meningoencephalitis (cerebral abscesses) - not common. Cerebral abcesses E. coli, S. aureus, Fusobacterium, Often in pituitary gland - goats have mass of capillaries surrounding pituitary gland. Also C. pyogenes - may extend from lymph nodes and abscesses of head and neck.

Meningoencephalitis/ brain abcesses

Signs: fever common, highly variable neuro signs depending on location - depression, incoordination, paraplegia, coma, head pressing, blindness

Also abnormal pupils, spasm of lower jaw, sound hypersensitivity, muscular spasms or rigidity, recumbency, opisthotonos, convulsions

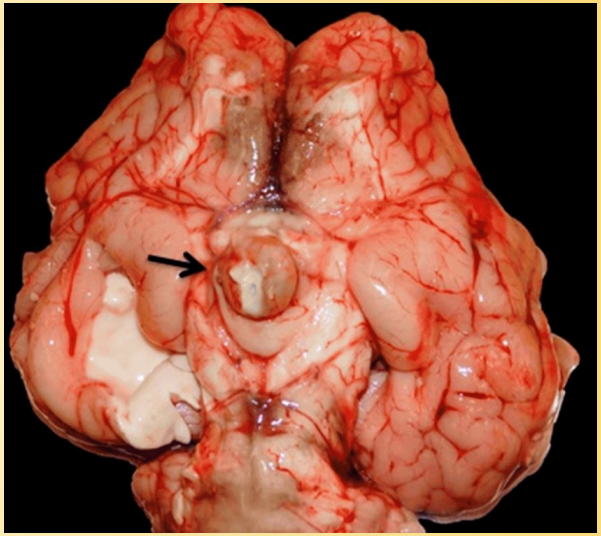


Photo Pituitary abscess in a calf - Stewart et al https://doi.org/10.3390/vetsci4010008

Meningoencephalitis - treatment

Guarded prognosis. But early treatment can be successful

1)Broad spectrum (bactericidal) antibiotics: Cephalosporins, IV/IM ampicillin, gentamicin, IV sodium penicillin G and/or Trimethoprim-sulfa continue for 48 hours after signs subside.

2) anti-inflammatories: Phenylbutazone 10 mg/kg SID, Flunixin meglamine 1-2 mg/kg BID im/iV, Aspirin 100 mg/kg PO BID

Dimly lit, quiet surroundings, if recumbent, turn frequently

Sometimes may need anticonvulsants/ sedation

Toxic plants, other toxins causing neurologic signs -

Ataxia is common sign with neurologic toxins

Chemicals Lead, salt, oxalate, urea, bromide, cyanide, nitrate,

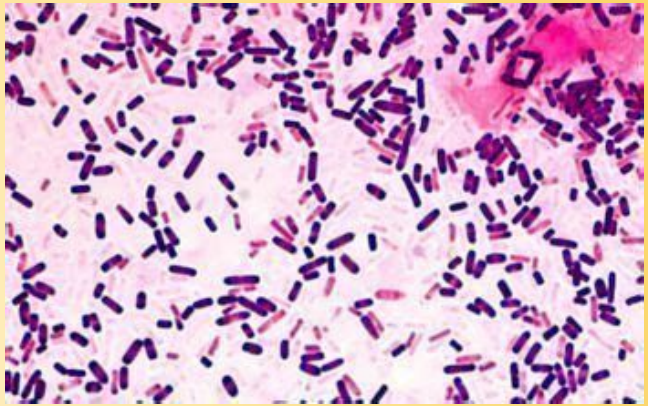
Insecticides: organophosphate, carbamate, chlorinated hydrocarbon

Diesel fuel

Drugs: Nitrofuran or levamisole overdose

Poisonous plants of the Indian Himalaya: an overview

Enterotoxemia



Clostridium perfringens bacteria (Gram + rods), spore forming

Types C and D being the most common forms Type D is found in older animals. Can be peracute, acute, and chronic. . Type C most commonly in lambs or kids younger than 3 weeks of age . *Changing conditions in the intestinal tract* may allow *Clostridium perfringens* organism to grow rapidly, releasing toxins.

Common in fast growing and well-conditioned animals grazing on lush pastures or rapidly growing feedlot lambs on high concentrate rations.

Enterotoxemia

Not primarily a neurologic disease, but may see sudden onset of neurologic signs:

Signs - Recumbancy, abdominal pain, convulsions, and sudden death. Death usually occurs within hours of onset of signs.

Kid goats are more likely to show signs of diarrhea before death.

Treatment is rarely effective (antitoxin, antibiotics), and prevention is far more likely to be successful.

Routine **vaccination** for *Clostridium perfringens* types C&D is very effective, Two doses - booster of the product 3 to 4 weeks after the initial vaccination

Often combined with tetanus toxoid.

Scrapie

Contagious, degenerative neurological disease of sheep and goats.
Prion - small protein based particle

Signs - Adult goats, 2-8 years of age. Slowly progressive (over 6 months) neurologic disease in adult goat. . Posture of carrying rear limbs forward, tail held up, ears forward. Restlessness, hypersensitive, bleating, increased muscle rigidity, hyperesthesia, head down, stomping, fine tremors.

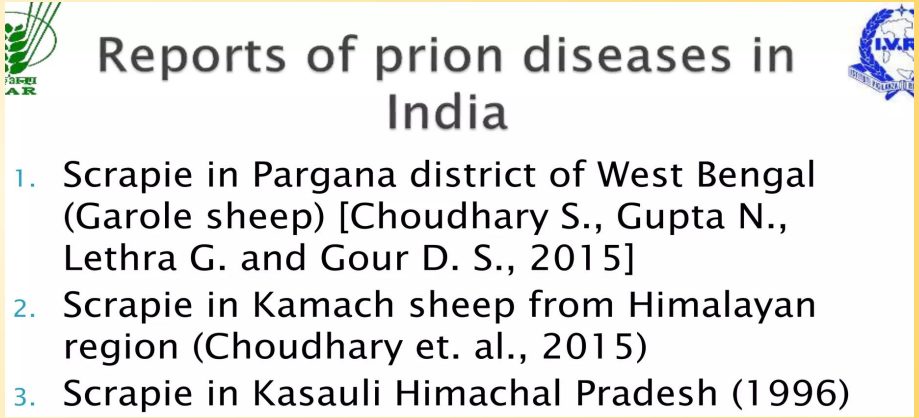
Pronounced incoordination, possibly teeth grinding, falling, listless, eventually anorexia and weight loss, pruritus,

Diagnosis - no lab tests exist. DDx for tremors and progressive incoordination - consider also CAE, pregnancy toxemia, copper deficiency, gid, spinal cord abscesses, rabies.

https://www.youtube.com/watch?v=Kkhak9yJ3H0

Scrapie

Not common disease, but no treatment or vaccination exists. Resistant to most methods of disease control such as disinfection, vaccination, chemotherapeutics. Euthanize if suspected based on signs



prion-diseases-in-india-spatial-and-temporal-distribution-

Rabies

Fatal to animals and humans. 20,000 human cases yearly in India, mainly transmitted from dogs.

Rhabdovirus. Spillover infections from wild reservoirs. Migration, changes in populations leads to increased incidence.

Susceptibility of goats depends on amount of inoculum into wound, number of nerves near wound, location of wound, and vaccination history.

VIrus spreads along peripheral nerves to CNS. Nonsuppurative encephalitis Incubation 2 weeks to several months. Shorter incubation if bite nearer CNS

Rabies

Signs: Depression or excitation, nystagmus, muscle spasms. Goats can become aggressive, attacking objects and handlers.

HInd limb proprioceptive deficit, ataxia, rear end paralysis, tail/penile paralysis, then ascending paralysis - finally recumbency, convulsions, death in 7-10 days.

Consider rabies in all neuro cases - so use gloves, etc. Avoid CSF contact in neuro cases

Diagnose by clinical signs, history, and animal death.

Brain and salivary glands submitted to authorities.(FA test of impression smears) Cool but do not freeze.

Vaccinate goats at 3 months, then annually.

Exposed animals: vaccinate at 0, 2, 4, 6 weeks. Quarantine 6 months.

Urea Toxicity

Urea -frequently included in ruminant diets to partially replace protein ingredients Highly toxic if consumed in excess. About 15-25 grams urea / 50 kg body weight in a single feeding in an unadapted or hungry goat will result in acute (ammonia) toxicity.

Urea from NPN compounds (eg, feed additives, fertilizers) - converted to **ammonia** in the gastrointestinal tract. Some ammonia is used by the rumen microorganisms together with carbohydrate for protein synthesis (High fiber diets lack enough carbohydrate for synthesis, increasing toxicity).

Ammonia in the GI tract diffuses into the bloodstream. The liver converts blood ammonia back to urea (urea cycle). Some excreted through the kidney, a portion passes back into the rumen via saliva.

If liver conversion capacity is overwhelmed- ammonia builds up to toxic levels in the bloodstream leading to **acute toxicosis**.

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Urea Toxicity Signs/ Diagnosis

Muscle and skin tremors, facial twitching, weakness, incoordination, frequent urination and defecation, blindness, recumbency, and death.

Some animals become bloated with abdominal pain, teeth grinding and loud bleating, dyspneic (from pulmonary edema) with frothy salivation , and hyperesthesia

As death nears, affected animals become cyanotic, anuric, and hyperthermic, with blood pH decreasing from 7.4 to 7.0. Increased blood and CSF ammonia concentration. Elevated PCV, K, P, and BUN.

Period from urea ingestion to onset of clinical signs - generally 30–90 minutes in goats/sheep. Death usually occurs within 4 hours Surviving animals recover in 12–24 hours with no apparent sequelae

Diagnosis Signs, and history of recent abrupt introduction of urea or other NPN suggests the diagnosis.

Urea toxicity - Treatment

- Infusion by stomach tube of **½ to 1 liter cold vinegar,** PO in goats, into the rumen.
- Oral **charcoal** (.5 kg PO) may also help decrease absorption. (Acetic acid lowers rumen pH , converting uncharged NH_3 to the charged ammonium ion (NH_4^+), which is less easily absorbed.
- One source (Pugh) suggests concomitant infusion of **iced (0–4°C) water.** This slows urease activity and dilutes the rumen contents
- **Supportive care**- IV fluid therapy (isotonic saline) to **correct dehydration** and IV calcium gluconate and magnesium solutions to relieve tetanic seizures. Convulsions may also be controlled with sodium pentobarbital or other injectable anesthetic agents.
- Administration may have to be **repeated** if clinical signs recur.

In severely affected valuable animals, emergency surgery to empty rumen. Replaced with a hay slurry, and a transfer of some rumen inoculum from a healthy animal

Urea Toxicity

Ruminal-reticular fluid specimens for analysis should be obtained before treatment.

Examination/ treatment for NPN poisoning may be difficult because of violent behavior. Animals that are recumbent and moribund usually do not respond favorably to treatment.

Prevention Urea should be added gradually to the diet. An adaptation period of at least three weeks is required for the animal to utilize urea efficiently.

Urea should supply no more than one-third of the total crude protein in forage or roughage-type diets and not more than one-half in the concentrate portion of the diet. (keep below 15g urea per 50kg BW/ day). Assure adequate carbohydrate.

Thank you!

Please contact me with any questions or comments

Paul Rudenberg DVM via vetnetfoundation@gmail.com

Sindh River Valley, Jammu and Kashmir