

Nutritional Disease and Problems in Sheep

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Metabolic disorders and nutritive deficiencies of sheep

1. Rumen flatulence

Causes:

- most often by eating legumes rich of protein (green alfalfa);
- animals are not accustomed to them.

2. Deficiency of vitamin E **and selenium**

Syndrome: pregnant ewes fed with a diet, which is poor of vitamin E and/or selenium often have lambs suffering of **„white muscle disease“**;

lambs: thin, pulse and EKG are irregular, cause of death is waxy muscle dystrophy in skeletal and heart muscles.

Treatment: supplement of selenium (Na-selenite per os and subcutan) and vitamin E for ewes and one-day old lambs

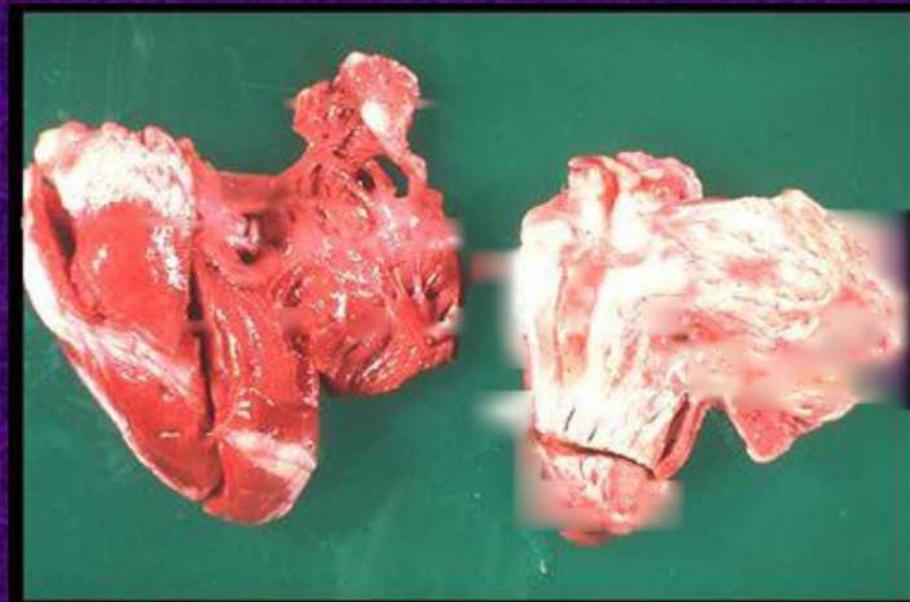
Symptoms

Skeletal

- Mild stiffness, obvious pain from walking, stiff gait, hunched appearance and inability to stand
- Lambs/kids tremble in pain when held in a standing position
- Normal appetites and bright personalities—until too weak to nurse
- Newborns born weak and unable to stand
- Sudden exercise may trigger WMD in older lambs or kids

Cardiac

- Shows signs similar to pneumonia
 - Difficult breathing
 - Frothy nasal discharge (may be blood stained)
 - Fever
- Heart and respiratory rates elevated and often irregular
- Both types may occur at the same time!



Prevention

- Deficiency occurs when animals are fed poor quality hay or straw or lack access to a pasture
 - Supplement the diet of the risk animals with Se or vitamin E
 - Especially pregnant animals since newborns are also at risk
- Sheep can get supplements either orally in their feed ration or through injections
- Goats can only get injections

3. White liver disease of lambs

- nutritional origin, mainly arises in spring;
- morbidity of 40%, mortality of 15%;
- acute form: 7-10 days; chronic form: 4 to 6 weeks;

Causes: deficiency of cobalt, that induces the deficiency of vitamin B₁₂;

In chronic case: propionate metabolism in liver is disturbed resulting accumulation of propionate;

- it might be accompanied by mycotoxin production.

3. White liver disease of lambs

Acute form: inflammation of eyelids and lips

originated in liver caused by secondary photosensitivity;

Chronic form: anaemia,

high weight loss, *laesios* on the ears, decrease of albumin, cobalt and vitamin B₁₂ of plasma.

- activity of AST, GGT and SDH significantly increase;

- degenerative fatty liver, necrosis of hepatocytes;

- haemosiderosis of spleen.

4. Thiamin deficiency of growing sheep

Syndrome: opisthotonus;

in the background: polyencephalomalacia (PEM) and cerebrocortical necrosis (CCN).

Occurance: generally in the age of 3 to 4 months.

Causes: high level of concentrate, no transition time (importance of preliminary feeding!);

In winter: hay containing antimetabolite of thiamine may induce it, number of thiamine-synthesizing bacteria is reduced in rumen.

4. Thiamin deficiency of growing sheep

Symptoms: in the first 2 to 5 days, excited behaviour, animals lay down and hold their head straight upwards („stargaizing”);

With no treatment: death in comatose status.

Similar symptoms: in listeriosis, ataxia caused by copper deficiency, scrapie.

Treatment:

- first dose of thiamin: 0.5-2.0 g/animal /day (50% intravenous, 50% subcutan);
- recovery dose: 0.1-0.5 g thiamin/animal

5. Enterotoxemia of fattening lambs

Other names: „overeating disease”, „pulpy kidney disease”

Occurance:

- suckling lambs;
- lambs fed with milk replacers;
- fattening lambs on high-concentrate diet;
- high-pregnant and lactating ewes.

Symptoms:

- pulpy kidneys (after necroctomy);
- toxins in rumen fluid.

5. Enterotoxemia of fattening lambs

Results:

- sudden death caused by toxin of type D (or sometimes C) produced by *Clostridium perfringens* (it occurs also in the intestinal tract of healthy sheep);
- in the case of feeding a diet rich in starch and sugar (concentrate, milk, fresh grass) *Clostridium perfringens* proliferates.

Prevention: vaccine containing toxoids.

Pregnant ewes: passive immunity for suckling lambs at age of 4-6 weeks; later: antiserum for lambs.

6. Acute lactic acid toxicity

Causes: high-concentrate diet with no preliminary feeding.

Results:

- concentration of lactic acid increases up to 2% in rumen fluid (normal: 50 mg%);
- pH in rumen: 4-4.2 (normal: pH 6-7);
- protozoa die in acid conditions in the rumen;
- high osmotic pressure develops (saliva, blood plasma);
- *collapsus* (preacute form), death.

6. Acute lactic acid toxicity

In acute and semi-acute forms: metabolic acidosis; increase of numbers of *Lactobacilli*, *Coli* and *Proteus* bacteria.

Symptoms: in the nervous system, small quantity of yellowish-green faeces, anuria, flatulence, breath smells very acidic.

Treatment: 20-50 ml of 5% NaOH solution intraruminally, reduction of inflammation, supply of thiamin.

Prevention: preliminary feeding of high-concentrate diet, feeding hay or straw before grazing.

7. Urolithiasis

Development of disease: split of *vesica urinaria*; urea flows into the abdominal cavity; „water belly”; death.

Occurance: castrated rams, fattening lambs on high-concentrate diet; increase of intake of Ca, P, Mg, K;

Grazing sheep: plants of high SiO₂-content.

Symptoms: sedimentation of minerals in urinary tract, painful and slow urinating; phosphate content of urine increases.

Prevention: decrease of P-intake (Ca:P = 2:1);

Acidification of urine (0.5% NH₄Cl in concentrate),

Drench of 7-14 g NH₄Cl for 3-5 days; in grazing sheep: 3-4% NaCl in diet, water: *ad libitum*.

8. Urea toxicosis

Background: feeding of NPN-compounds to fattening lambs with no preliminary feeding;

Toxic level of urea: 0.4 g per kg body weight.

Treatment: drinking of 500 ml household vinegar (20%) diluted 10-20 times;

Intravenous injection of maleic acid containing glucose (0.5ml per kg body weight).

9. Pregnancy toxicosis in ewes (sheep ketosis)

Occurance: high-pregnant ewes.

Background: insufficient quantity of concentrate in diet; transitional glucose deficiency, energy eventually supplied from fat depots; pregnancy toxicosis (ketosis); long period of *hypoglucaemia* may cause damages in brain tissues.

Symptoms: unstable movement of high-pregnant ewes (faltering steps); comatose condition before death.

Necroctomy shows: healthy foetus(es) died before the ewe's death; yellow liver with rounded edges.

10. Milk fever in ewes

- a. Classical (Ca ↑)
- b. Not typical (P ↑)

Syndrome: see in dairy cow

Milk Fever

- Ewes vs. Dairy Cows
- Cause
 - Calcium deficiency ??
 - Stress induced

Milk Fever

- Late gestation ewes carrying triplets
- Symptoms
 - Depressed, lethargic, recumbent
- Treatment
 - Calcium gluconate
 - Rapid response

Copper Toxicosis

- Breed Susceptibility
- Mineral interactions-Mo & Su,
 - along with high Zn & Ca
- Normal copper, low molybdenum

Copper Toxicosis

- Prevention
 - Sheep specific feeds
 - No additional copper
 - Feed some Mo
- Copper & Molybdenum
- are both toxic

Pregnancy Toxemia

- Cause - Excess fat catabolism and ketone accumulation
- Prevention
 - Over conditioned ewes
 - Increased conc. feeding LG
 - Fetal scans
 - Pre-lambing shearing

Pregnancy Toxemia

- Treatment
 - Propylene glycol
 - Induced parturition
- Severe cases usually do not recover

Grass Tetany

- Hypomagnesemia
- Cause: excess potassium
- Lactating ewes on lush, spring pasture
- N fertilization increases risks

Grass Tetany

- Magnesium oxide in mineral
- poor palatability.
- Sudden death-confirmation by necropsy
- Stress induced