University of Mosul
Lecture No.: 5
College of Veterinary Medicine
Date: 2025
Unit of Scientific Affairs
Website:

Lecture title: **Nonprotein nitrogen toxicosis (urea toxicosis)**

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Summary:

••Urea is a common, inexpensive, and readily available form of nonprotein nitrogen (NPN) used in ruminant rations and as a fertilizer.

•Urea is exists as food supplement, solid mineral blocks, in liquid products such as molasses, or used as a fertilizer.

•Toxicosis occurs when cattle or sheep gain access to large amounts of NPN, are fed larger quantities than they are accustomed to, ingest improperly mixed feeds, or drink polluted water.

Epidemiology

Occurrence

•Urea is used in agriculture as a feed additive for ruminants to provide an inexpensive protein substitute in the diet and as a fertilizer on crops, pastures, and fields.

•Natural occurring urease in the rumen supports the hydrolysis of urea to ammonia

Animal Risk Factors

•Ruminants are more able to assimilate ammonia into protein when adequate amounts of readily available carbohydrates are provided

•In the absence of sufficient digestible carbohydrate, such as when only roughages are fed, urea is more toxic.

•The toxic dose of NPN in ruminants depends on a number of factors such as current diet, acclimation to product, rumen pH, body temperature, and the presence of other diseases.

•Urea needs to be gradually introduced into the diet over several days to allow the rumen bacteria time to become accustomed to the ammonia source.

• in cattle of 0.44 g/kg BW produce signs of poisoning within 10 minutes and dose rates of 1 to 1.5 g/kg BW are associated with death.

• **Sheep:** A dose rate of 2 g/kg BW is quickly fatal.

• **Horses:** poisoning rarely occurs.

Pathogenesis

•Under normal circumstances, urea in the rumen is broken down by urease to ammonia, which is then used by rumen bacteria to synthesize proteins.

•Excess ammonia remains in the rumen as the ionized ammonium ion.

•The sudden introduction of large amounts of urea upsets this reaction and toxicosis occurs. absorbed across the rumen into the systemic circulation.

•Rumen pH is elevated and more ammonia remains in the nonionized form and is absorbed.

•The onset of signs occurs in 10 to 30 minutes after feeding.

•The severity of signs is directly related to blood ammonia levels and not to ammonia levels in the rumen.

•Excess blood ammonia interferes with energy metabolism, inhibits the citric acid cycle, and results in systemic lactic acidosis.

•Hyperkalemia, associated with systemic metabolic acidosis, may cause cardiac arrhythmias and arrest.

Clinical findings

Cattle and Sheep

•Signs of toxicosis occur as early as 10 minutes after the urea is eaten and include :

1-Severe abdominal pain, frothing at the mouth and nose

2-bellowing, hypersensitivity to sound,

3-movement aggressive,

4-muscle tremors, incoordination, weakness, dyspnea, bloat, and violent struggling.

5-Less severe cases are drowsy and recumbent.

6-In severe cases death occurs in a few minutes, but more commonly, animals die about 4 hours after ingestion.

Diagnosis

1-history 2-clinical signs 3-Clinical pathology

1-Signs are visible when rumen ingesta levels of ammonia are 1000 mg/L

2- serum levels of ammonia nitrogen (NH3-N) are 10 to 13 mmol/L, and when blood ammonia nitrogen concentrations reach 0.7 to 0.8 mg/ dL. the serum ammonia levels (up to 1719 µmol/L).

3-the higher the blood lactate levels (up to 26.01 µmol/L) were in a group of poisoned steers.

4- Blood pH (7.24) in this group was consistent with metabolic acidosis.

4-Necropsy findings

There are no characteristic lesions at necropsy, but most animals show generalized congestion, hemorrhages, and pulmonary edema.

Differential diagnosis :

• Acute bovine pulmonary emphysema and edema

• Acute hepatic insufficiency • Acute salt toxicosis • Anaphylaxis

• Hypomagnesemia

T**reatment**

1-5% acetic acid (vinegar) (0.5–1 L orally to a sheep, 4 L orally to a cow) .

2-Cold water (10–30 L PO for adult cattle, repeat as needed).

•No primary treatment is likely to be effective, especially in large herd situations, because the mortality rate is high and death occurs before treatment can be instituted.

•Cold water will dilute excess urea, temporarily lower rumen pH, and slow NPN metabolism by urease enzymes.

•The only really effective treatment is prompt and efficient emptying of the rumen, either via a large-bore stomach tube or by Rumenotomy.

control

•Urea is highly toxic and care is essential when handling it in the vicinity of animals.

•Feed manufacturers' recommendations about maximum concentration of urea in prepared rations .

•In dairy cows, urea should be fed at 1% of the concentrate, 135 g per animal per day, and not more than 20% of the total crude protein (including other NPN sources).