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Popular Article

Urea and ammonia poisoning in ruminants: A brief overview

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Pasture and crop fields, urea is used as a fertilizer. In snow-covered residential areas, itis occasionally used as a substitute for common salt to melt snow and ice. However, during World War II, it began to be used as a low-cost source of non-protein nitrogen (NPN) in the diet of ruminants. When combined with enough other feeds, urea is significantly more tolerable. Urea can be added to the whole feed (on a dry weight basis) in amounts of 2 to 3 percent without causing toxicity.

Ruminants adapt to eating on urea quickly. Since it has been discovered that cattle may assimilate even 400 g of urea per day with adequate adaptation, but even as little as 50 g/day is poisonous without proper adaptation, animals should become acclimated to urea feeding by progressively increasing its level in the ration/feed.

The pace of ammonia synthesis and absorption affects the urea's toxicity. All mammalian species can become poisoned by ammonia, although ruminants are the most vulnerable. Horses (equines), where urea hydrolysis occurs in the caecum, appear to be more resistant to relatively high doses of urea than ruminants. Pigs, however, are quite resistant to urea, even in very high doses. This is most likely because ruminants have a larger ability thannon-ruminants to synthesize urea in the liver or have higher glutamine synthetase activity in the spleen, liver, and brain.

Oral LD., value of urea in cattle and sheep is 1.0-1.5 g/kg and horse is 4.0 g/kg while toxic

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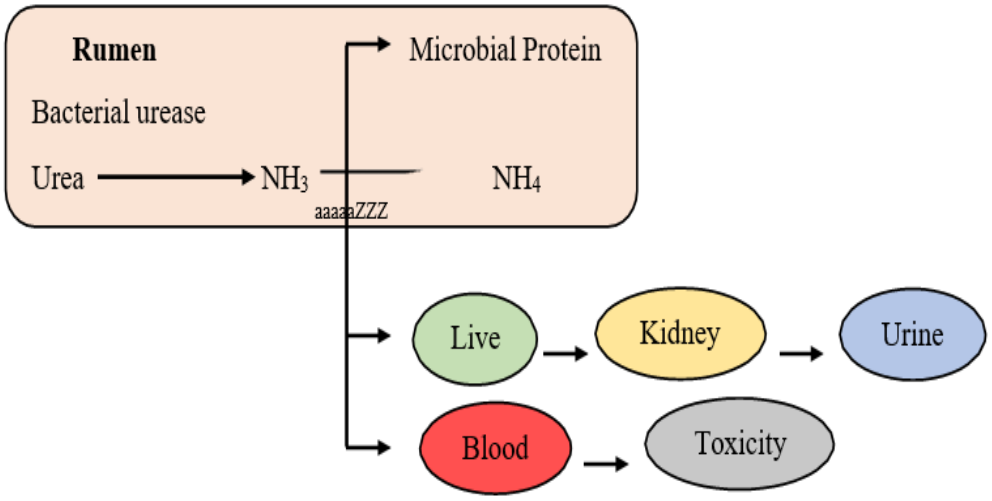
dose in cattle and sheep is 0.3 -0.5g/kg.

Sources of poisoning

accidentally providing huge amounts of NPN urea-molasses feed to unfamiliar animals, poorly mixing feed, or accidentally ingesting solid or liquid form of urea due to improper storage or spillage.

Mechanism of Toxicity

In the rumen, urea is hydrolyzed. The majority of the ammonia produced from the rumen is in the form of ammonium ions (NH_4^+), which are highly water soluble and rarely absorbed, meaning that low pH encourages NH_4^+ production. As absorbed ammonia is often included in the urea cycle and eliminated as urea in urine as shown in figure, the body detoxifiesammonia if



blood ammonia absorption is within specified limitations. The liver is the organ where, ammonia (NH_3) is converted to urea. Due to their increased hepatic urea-synthesizing capacity, ruminants are better able to handle ingested NH_3 than non-ruminant species.

When concentration of NH_3, in rumen exceeds 80 mg/dl, NH_3, appears in the peripheralblood and high NH_3, concentrations are built up in blood stream and thus NH_3, accumulates intissue cells. When blood NH_3, nitrogen reaches 0.80 -1.30 mg/dl, clinical signs of poisoning become apparent.

Clinical signs

Clinical symptoms and their severity differ from animal to animal, and occasionally the animals themselves are discovered dead. Depending on how quickly ammonia is produced in

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the rumen and absorbed into the bloodstream, the onset of the symptoms may be rapid or delayed. However, the most severe symptoms appear first, and death usually happens between 30 minutes and 4 hours after intake. Poisoning is characterised by weakness, early agitation, salivation, frothing at the mouth and nose, teeth grinding, abdominal pain, bradycardia, a prominent jugular pulse, dyspnoea, bloating, forced quick breathing, and weakening and slowing of the pulse and respiration. When the blood ammonia content is above 5 mg/dl, there will typically be severe groaning, shivering, twitching of the ids, lips, and tail, ataxia, final tonic convulsion, and death following fierce struggling and bellowing. There isn't any weird stance or jumping on the hidden things, though. Between convulsions, animals seem rigid rather than dejected. Although colic is a notable symptom.

Post-mortem lesions

No characteristic post-mortem lesions are present in urea poisoning, however, some of the commonly observed lesions are as follows:

I. Ammonia odour in the rumen.
II. Generalized, congestion, haemorrhages and vascular injuries.
III. Hydropericardium and hydrothorax are present.
IV. Haemorrhagic enteritis with oedema and ulceration of intestinal mucous membrane.
V. Liver is pale, enlarge and friable.
VI. Haemorrhagic degenerative changes found in the brain.
VII. Fatty degenerations occur in liver and kidneys.
VIII. Pulmonary oedema and acute catarrhal bronchitis, peribronchial and intra alveolarhaemorrhages.
IX. Pigs can get encephalomalacia that is comparable to salt poisoning, but without theeosinophilic aggregation.

Diagnosis

I. History of urea feeding of intake
II. Clinical signs
III. Post-mortem lesions
IV. Laboratory tests show elevated levels of ruminal fluid and blood ammonia, ruminal pH greater than 7.5, and elevated blood urea nitrogen. Feed analysis for urea, ammonia salt etc.

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V. Stomach / ruminal contents for urea of ammoniacal fertilization.

Differential diagnosis

X. Haemorrhagic enteritis with oedema and ulceration of intestinal mucous membrane.

XI. Liver is pale, enlarge and friable.

XII. Haemorrhagic degenerative changes found in the brain.

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VIII. Post-mortem lesions

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Differential diagnosis

I. Arsenic poisoning.

II. Lead poisoning.

III. Organochlorine pesticides Stomach/ruminal contents for urea or ammoniacalfertilization.

IV. Nitrate and cyanide poisoning

V. Encephalitic disease, enterotoxaemia, brain engorgement.

VI. Strong caustics poisoning.

VII. Organophosphate toxicity

Treatment

Urea poisoning has no specific therapy, and there is no effective antidote either. Eliminate the exposure source first. The goal of treatment is to lower the level of ammonia in the blood, either by reducing ammonia production or by accelerating the conversion of

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ammonia to urea. Weak acids are typically supplied by mixing enough cold water with vinegar or 5% acetic acid. Cattle receive 4.0 litres of acetic acid, whereas sheep receive between 0.5 and 1.0 litres. In addition to diluting the ruminal contents, it also slows the rate of urea hydrolysis by reducing urease activity, lowers ammonia synthesis by lowering rumen pH, and stimulates diuresis. By using a trocar and canula, extra gas that has built up in the rumen can be evacuated. The most efficient form of treatment, nevertheless, is rumenotomy or stomach tube rumen emptying. The other course of treatment typically focuses on symptoms.

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