

INDIGESTION DUE TO ACUTE RUMEN ACIDOSIS

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Hydrochloric acidosis (abomasum reflux)

Cours_ pathology of farm animals.
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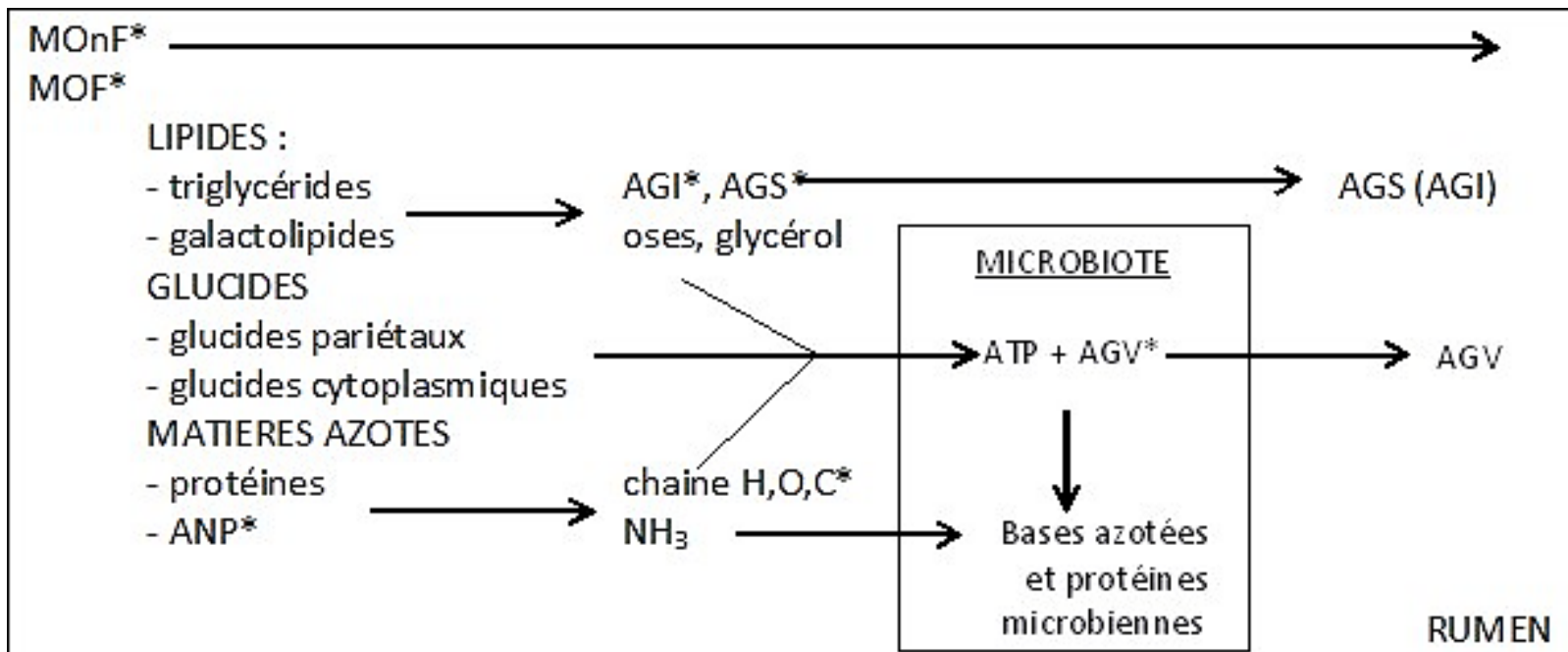
Introduction

- The rumen represents an ecosystem, within which changes in environmental conditions are at the origin of variations in the composition of the bacterial flora.
- variations that can be the cause of significant pathologies
- A symbiosis exists between the ruminant and its rumen bacterial population,

- the digestive system of the ruminant has particularities that allow it to digest the plants they ingest.
- Depending on the nature of the fermented carbohydrate, there are two types of bacteria.
- 1- Amylolytic bacteria, whose preferred substrate is starch, synthesize propionate and prefer pH under 6.
- 2- Fibrolytic bacteria mainly attack wall carbohydrates to give acetate and butyrate, and like pH above 6.

LA DIGESTION RUMINALE DES ALIMENTS;

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MO nF : Matière **O**rganique **n**on Fermentée.
MO F : Matière **O**rganique **F**ermentée.

ANP : Azote **N**on Protéique.

AGS : Acides **G**ras **S**aturés. **AGI :** Acides **G**ras **I**nsaturés.

AGV : Acides **G**ras **V**olatils.

- The rumen includes a dense and diverse population of microbes encompassing the three domains of life: bacteria, archaea, eukaryotes, plus viruses, mainly phages.
- Bacteria are predominant, with 10^{10} cells per g of rumen content and cover most of the metabolic functions existing in the rumen.
- Eukaryotes include protozoa (10^6 cells per g of rumen content), which represent 30 to 50% of the microbial biomass of the rumen
- Mushrooms (10^5) (champignons!)

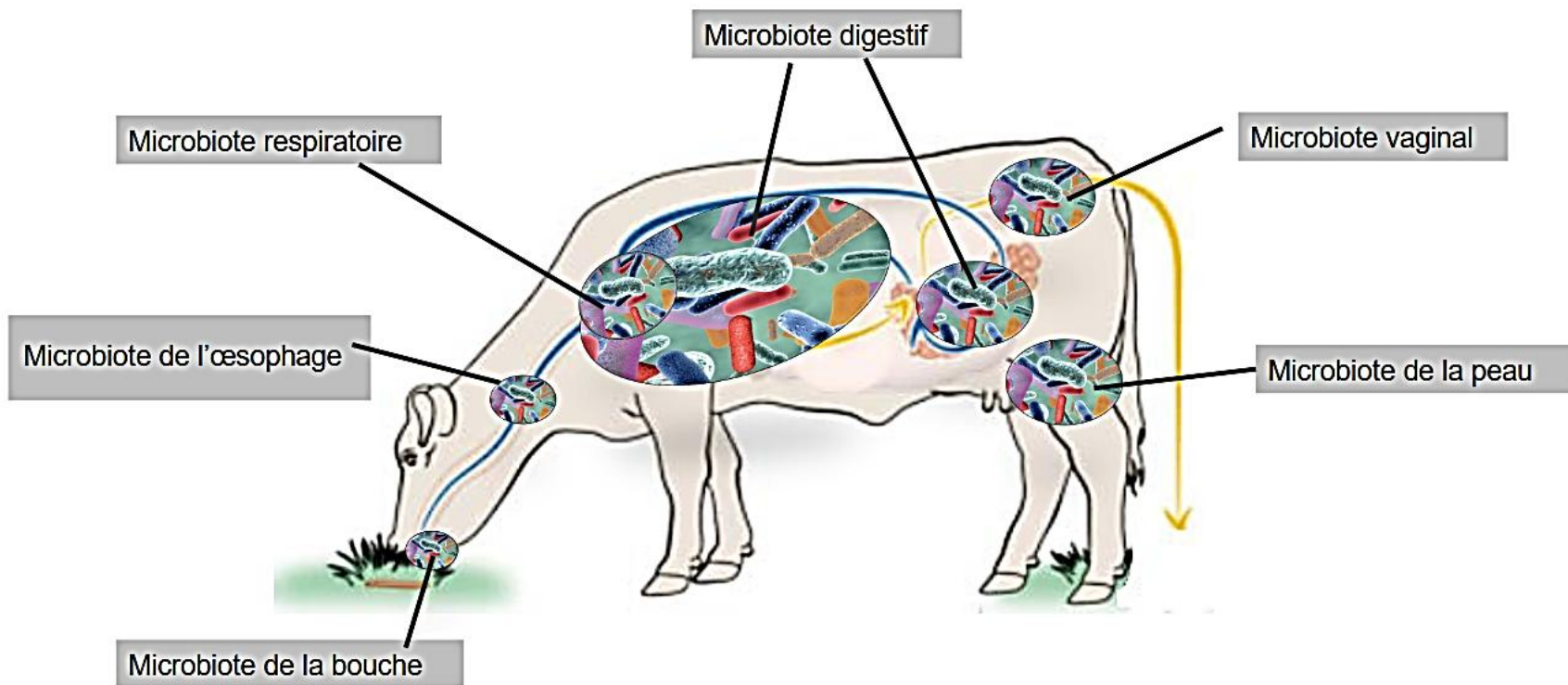
Importance of microflora

"ALL THE RUMINANT IS IN ITS BELLY"

Feeding a ruminant = means feeding a dense
and diverse microflora

in Franch: "tout le ruminant est dans sa
panse)"

Main microbiota of ruminants (different bacterial phyla)



Metabolic Results:

Volatile fatty acids (VFAs) which are a source of energy.

PDIM: Digestible Protein in the Gut of Microbial Origin

B-complex vitamins

This digestion requires:

✓

✓ Stagnation of the feed for 24-48 hours,

✓ Rumen stirring movement

✓ Temperature of 39.5°C

✓ Anaerobic

✓ Humidity 80-85%

✓ pH= 6-7

- MAT: Total Nitrogenous Matter which corresponds to the crude protein content. It is obtained by multiplying the nitrogen content by a coefficient of 6.25. Digestibility and the nature of the amino acids that make up proteins are not taken into account.
- - MAD: digestible nitrogenous matter. It is the MAT minus the nitrogen that is found in the faeces, so it is not used and not transformed in the digestive tract.
- - PDI: Digestible Proteins in the Intestine.
- - PDIA: PDI from dietary proteins that are not broken down in the rumen.
- - PDIM: PDI of microbial origin. A distinction is made between rumen-degraded nitrogen (rumen-degraded nitrogen) and rumen-fermentable energy-enabled IMDPs (IMDPs).
- - DINPs = DIAD + IMDP (related only to total nitrogen content = MAT).
- - PDIE = PDIA + MIDP (depends on nitrogen content and energy value).

Carbohydrate digestion

- Production of volatile fatty acids:
- Acetic acids (C2)
- Propionic acids (C3)
- Butyric acids (C4)
- Proportion varies with the intra-ruminal pH and therefore the rate of fermentation which depends on the nature of the carbohydrates and their physical presentation and the splitting of the intake
- Forages lead to acetic fermentation favourable to the butyrate rate (beurre)
- Starchy concentrates promote the formation of propionic acid beneficial to the protein level (cheese)

Acute rumen acidosis

- Acidosis is induced by rations rich in concentrates or other products from the manufacture of sugars (molasses) rich in easily degradable sugar)
- This metabolic disease causes dysbiosis characterized by a decrease in the richness and diversity of the ruminal and even intestinal microbiota.
- it results in a digestive disturbance causing the pH to drop below 5.

Acute rumen acidosis

Dietary causes:

Sudden change in diet, especially at the beginning of lactation (first 2-3 weeks)

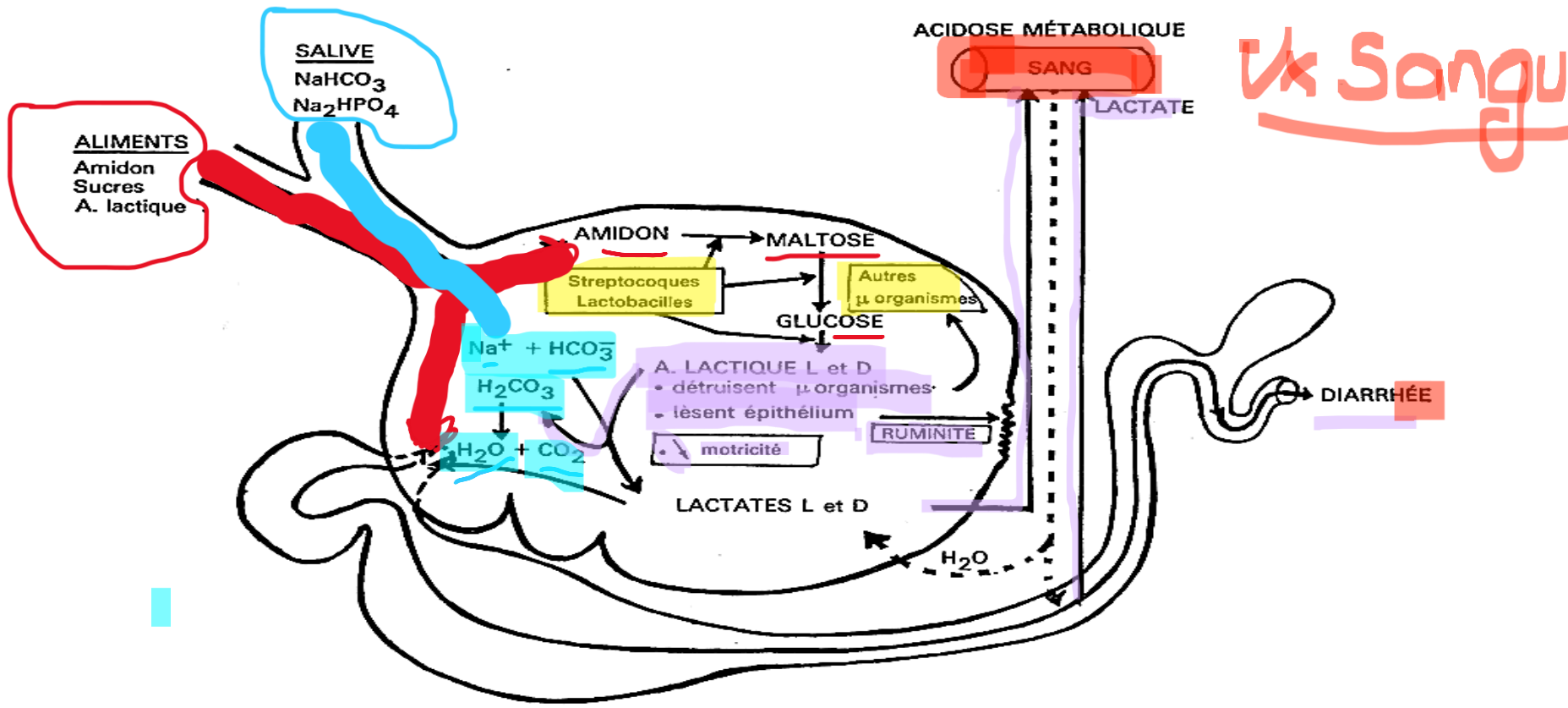
Lack of ballast: < 40% DM fodder and < 1kg long hay; crude fibre < 15%

Excess fermentable carbohydrates: starches + sugars 25-30% DM;

Rapidly fermentable carbohydrates < 35-40% DM

Poor distribution of concentrates during the day (exclusive distribution outside the basic ration)

Pathogénie de l'acidose lactique



Clinical signs of acute acidosis

- Three clinical entities can be distinguished in acute acidosis:
- a shock syndrome
- metabolic acidosis
- digestive disorders



Clinical symptoms

1. Shock syndrome:
2. Hypovolemic shock:
3. increase of ruminal osmolarity, by ruminal accumulation of the two isomers of lactic acid.
4. Water leaves the connective tissues (the extracellular and intracellular sectors) to accumulate in the rumen.
5. What causes peripheral dehydration: positive skin fold test and enophthalmia
6. The "loss" of body water corresponds to 8% of the body weight, of which 60% is of extracellular origin
7. symptoms of depression (refusal to eat, PICA), tachycardia, low blood pressure, anuria, decubitus,



Regurgitation of rumen substances through the nostrils (1), dung made up of ingested wheat seed (2) and dung of diarrhoeal consistencies (photos Dahmani Ali)



2. Metabolic acidosis:
3. hyperlactacidemia:
4. by absorption via the rumen and intestines. the pH of the blood is reduced from 7.4 to 7.2
5. decrease in alkaline reserve:
6. increased density of urine with a pH of 5 (instead of 8), and
7. lactatory,
8. - the haematocrit has been increased from 30% to 35%.
9. - Decreased blood pH from 7.4 to 7.2
10. - reduction of bicarbonates from 25 to 15 mEq/l
11. Endotoxin resorption:
12. (acidophilic bacteria)
13. laminitis Sometimes (consequences if healing)
14. tetan-like symptoms such as hyperesthesia, teeth grinding, chewing and locomotor disorders

Clinical signs of acute acidosis



dung of diarrhoeal consistencies (photo Dahmani Ali)

Clinical signs of acute acidosis

3. Digestive disorders:
4. Stopping the motor skills of the forestomachs:
5. related to the activation of epithelial chemoreceptors located in the rumen wall. when volatile fatty acid levels exceed a certain threshold, stimulation of these receptors leads to central inhibition of motor skills;
6. the hollow of the left flank is distended with a pasty sensation,
7. elastic on external or transrectal palpation;
8. On auscultation, there is disappearance of contractions with crackling noises.
9. The rumen pH at this time is between 4 and 5

- osmotic diarrhea:
- The demand for water associated with the accumulation of lactate in the rumen on the one hand, and the passage of lactic acid and carbohydrates into the intestinal lumen on the other hand, the latter can be complicated by a bacterial entity

Symptoms / Acute form

- The high concentration of undissociated VFAs in the rumen and blood is characterized by gastrointestinal atony
- The resorption of a large quantity of lactic acid D results in depression and nervous disorders due to damage to the nerve centres.
- The intake of water in the rumen results in:
 - lowering of diuresis,
 - decreased blood pressure
 - tachycardia and rapid breathing at the beginning.

- The passage of water through the rumen will lead to extracellular dehydration and then
- with lowering of diuresis, a decrease in blood pressure with
- tachycardia and respiratory acceleration (polypnea).
- Dehydration increases the density of urine and results in hemoconcentration.
- Later, the pH of the blood drops below 7.30, the transport of CO₂ is decreased and it accumulates in the tissues. There is cellular anoxia.
- Nerve cells are the most sensitive.
- These facts explain the adynamia or even coma of animals.

Symptoms / Chronic form:

- There is an orientation of the metabolism towards the production of C3 (propionic and lactic acid) with increased papillae, agglutination and detachment.
- We move from hyperkeratosis to parakeratosis and ulcers.
- Liver abscesses are explained by the passage of germs through the injured wall of the rumen.
- There is a decrease in the fat content of milk due to the decrease in the production of VFAs
- C2 and C4.
- Atony can lead to a displacement of the abomasum or twisting of the cecum.
- Hyperlactacidemia results in chronic laminitis in the podophyllum.
- The frequency of diseases is increasing, such as enterotoxemia and
- Fat cow (obesity)

Treatment of acute acidosis

- Emergency treatment is required to:
- Correct intraruminal acidosis and prevent lactic acid production:
- In severe cases (at the beginning of the course): a rumenotomy is indicated.
- The rumen is emptied, cleaned, and the rumen juice (10 to 20 liters) is added, with a few handfuls of hay.
- Less severe (rumen lavage): A rubber tube 25 to 28 mm in diameter is introduced into the rumen, and hot water is pumped inside until a distension of the left paralumbar fossa is observed, at which point the rumen is then able to empty itself thanks to gravity. The rumen can be completely drained after 10 – 15 irrigations

Traitement de l'acidose aiguë

- Failing this, alkalizing substances (magnesium carbonate or magnesium hydroxide) are administered orally. The initial doses, of 1 g/kg live weight, may be followed by lower doses repeated at intervals of 6 and 12 hours

Treatment of acute acidosis

- 2. Restore hydroelectric balance and maintain blood volume: Systemic acidosis is treated by intravenous intake of Intravenous Sodium Bicarbonate 5% at a dose of 5 liters for a 450 kg animal,
- for the correction of systemic acidosis This infusion is followed by that of an isotonic sodium bicarbonate solution (1.3%), at a dose of 150 ml/kg body weight over the following 6-12 hours.

Treatment of acute acidosis

- 3. Restore bacterial flora,
- restoring rumen motricity (transfaunation): a loss of the cellulolytic population is corrected by using rumen juice from healthy animals (from slaughterhouses, or a regurgitated bolus from a live animal); The rumen content thus withdrawn is administered by drenchage, or deposited directly in the rumen after rumen emptying if a rumenotomy has been performed

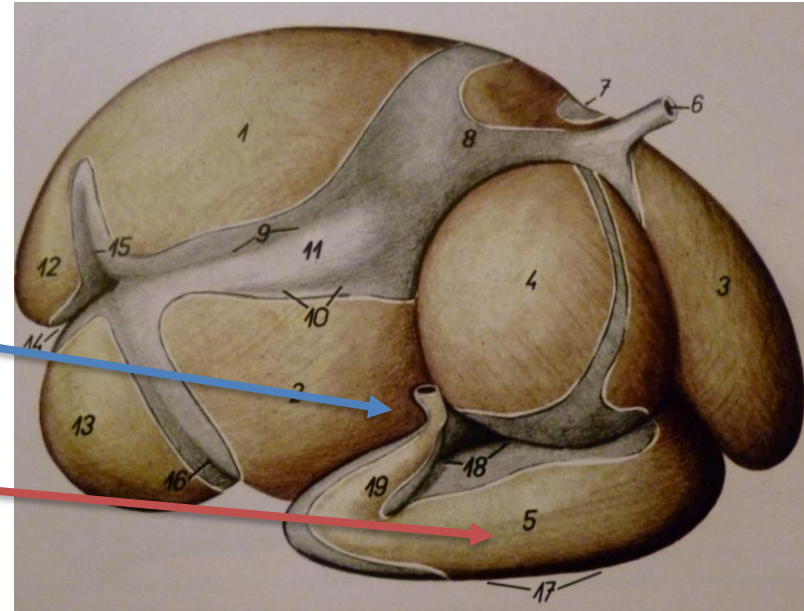
Traitement de l'acidose aiguë

4. lutter contre les complications:

- l'utilisation d'antihistaminique (pour prévenir la fourbure)
- parasymphomimétiques pour stimuler la motricité ruménale
- de thiamine ou de levure pour stimuler le métabolisme de l'acide lactique
- antibiothérapie: lutter contre les surinfections

Hydrochloric acidosis (abomasum reflux)

- Hydrochloric acidosis of the rumen is related to generalized gastrointestinal stasis or obstruction (Hoflund syndrome: by stopping or slowing down at the pyloric orifice)
- The obstruction does not allow the passage of chyme from the abomasum to the intestine and leads to a reflux to the rumen of the omasal contents rich in hydrochloric acid



Pathogenesis

- The hydrochloric acid of abomasum reflux corresponds to acid lost, not recoverable in the intestine, for plasma and this results in metabolic blood alkalosis with hypochloremia (50 instead of 95 mEq/l)
- This is accompanied by hypokalemia (2 instead of 4-5 mEq/l) due to anorexia
- in cattle with hypokalemia associated with metabolic alkalosis, aciduria (renal reabsorption of Na^+ in exchange for an H^+ ion) is observed

Pronostic et traitement

- Il est sombre
- Il consiste en l'apport de laxatifs (hydroxyde de Mg : 0,5 à 1 kg/jour
- perfusez au gluconate de Ca pour augmenter la motricité abomasale
- administrer en intraveineux des solutions riches en NaCl en KCl (8 à 10 litres de solution de NaCl à 0,9 %, 4 litres de solution de KCl à 1 %, en ne dépassant pas 3 mEq/kg de poids vif
- Après ce traitement d'urgence, on pourra pratiquer une ruminotomie lors d'obstruction partielle de l'abomasum afin d'injecter directement dans la cavité abomasale du sulfate de Mg.
- Cette ruminotomie permet également un massage de la caillette

Thank you