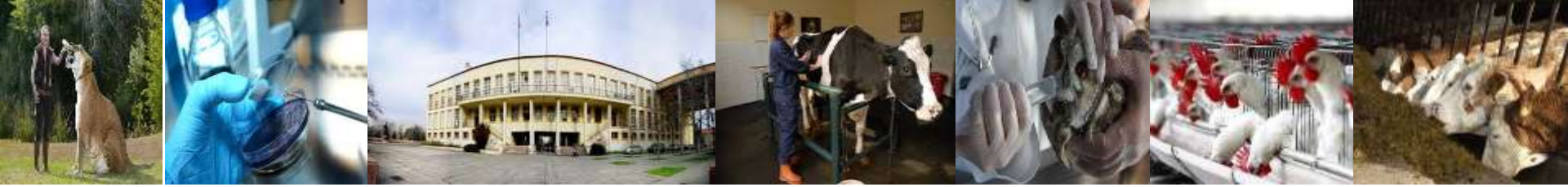

Ketosis

Assoc. Prof. Özge SIZMAZ

University of Ankara Faculty of Veterinary Medicine Department of Animal Nutrition and Nutritional Diseases, Ankara, Turkey

Introduction

- Ethiology
- Type of ketosis
- Sympthoms
- Diagnosis
- Treatments and protection

- Pregnancy toxemia

Ketosis = Acetonemia

- Energy metabolism disorder
- 10 d and 6th week after calving (esp. 3rd week)
- Holstein cow < Jersey cow (insidence)

Ethiology

- 1. Primer Ketosis
- 2. Seconder Ketosis
- 3. Alimentary Ketosis
- 4. Spontenous Ketosis

Primer Ketosis

- Negative Energy Balance

  - stress factor → DMI abatement

↓

Metabolic unbalance

↓

Clinical Ketosis

Seconder Ketosis

- Diseases that are caused to reduce DMI

- Deplaced abomasum
- Reticuloperitonitis

Alimentary Ketosis

- By consuming fermentated feeds that contain highly ketogenic components.

- Butyric acid
- Acetoacetic acid (AAA)
- BHBA

Maize silage and ketosis relation????

Spontaneous Ketosis

- Unknown reasons
- Enough and balanced feeding

- Generally ketosis forms in Winter

Ethiology

Briefly;

1. Insufficient exercise, excessive lubrication at calving and insufficient energy intake in first lactation period.

Insufficient energy intake; using fat reserve and protein

CHO = VFA …

Propionic acid → Oxaloacetic acid → glycose → TCA (crebs)Cycle → energy source

Ethiology

- Activated Acetyl Co A is formed by acetic and butyric a.
- When glycose metabolism contuniue their function, fats are formed by activated acetate
- and if there are sufficient oxaloacetate, activated Co A join TCA, degrade, used for energy source.

Ethiology

- If there are insufficient OA, two molecules of Acetyl Co A link = acetoacetic acid.

BHBA and Acetone

Ketone bodies

Ethiology

2. Silages that include high level of butyric acid
3. High level of CP in feed → rumen → butyric acid
4. Starvation
5. Hepatic failure
6. Co insufficiency
7. Excessive feding
8. Genetic susceptibility

Symptoms

- 2-4 d before clinical symptoms, DMI decrease, milk production reduce ====== acetone odour in breath.

- Progressive of disease; solid, dark color and sticky feces
- Mortality is not observed but, fatty liver and liver degeneration are observed
- Fever is a marker for seconder ketosis.

Diagnosis

- Odour (Acetone)
- Milk fat / milk protein ⬆ ======= early diagnosis
- Blood BHBA level

  -1 mmol/dl ⬇ normal (healty cow)

  -1- 1,4 subclinical ketosis

  - 1,4 ⬆ clinical ketosis

Treatment

Care of 3 points for succesfull treatment

1. Blood glucose level should be brought to normal level quickly
2. OA level should be increased in liver.
3. Application should be made to increase the evaluation of glycogen precursors especially propionic acid from the ration

Treatment and Protection

Glucose solution (40%), iv, 500 ml

Propilane glucole (a glycogenic material), oral, 150 ml (2 times/d; 3-4 d)

Add Co

Glycocorticoids (increase the evaluation of glycogenic materials in liver)

Protection

1. Sufficient energy intake after calving
2. BCS in dry period (3,5), prevent lubrication
3. Bad quality silage X
4. Before and after calving, 2-3% propylene glycole to diets

Protection

5. Last 2-3 w of dry period and first 10 w following calving==== niasin (6-12 g)

6. Exercise to the cow, staying in closed ban

7. Add Co, P and Iodine to diets

Ketosis in Sheeps (Pregnancy toxemi in Ewe)

Sheep with twin, triplet lambs

and 5th months of pregnancy

Ethiology

-insufficient feed intake

-instant feed change

-bad feed management

-starvation

Ketosis in Sheeps (Pregnancy toxemia in Ewe)

Mechanism

If the glucose secreted by the mother cannot meet the need for fetus, ketosis occurs

Need for glucose = from fatty a. & amino a.

Long time hypoglycemia --- supresses insülin production & increase mobilisation of f.a.

↓

Insufficient OA in TCA → Co-A reverse to ketone bodies