Pregnancy Toxaemia in sheep/goat Ovine ketosis



Introduction

- It is highly fatal disease occurring in late pregnancy characterized by Hypoglycaemia, ketoneaemia and low liver glycogen content.
- Negative energy balance and multiple fetuses predispose the disease.

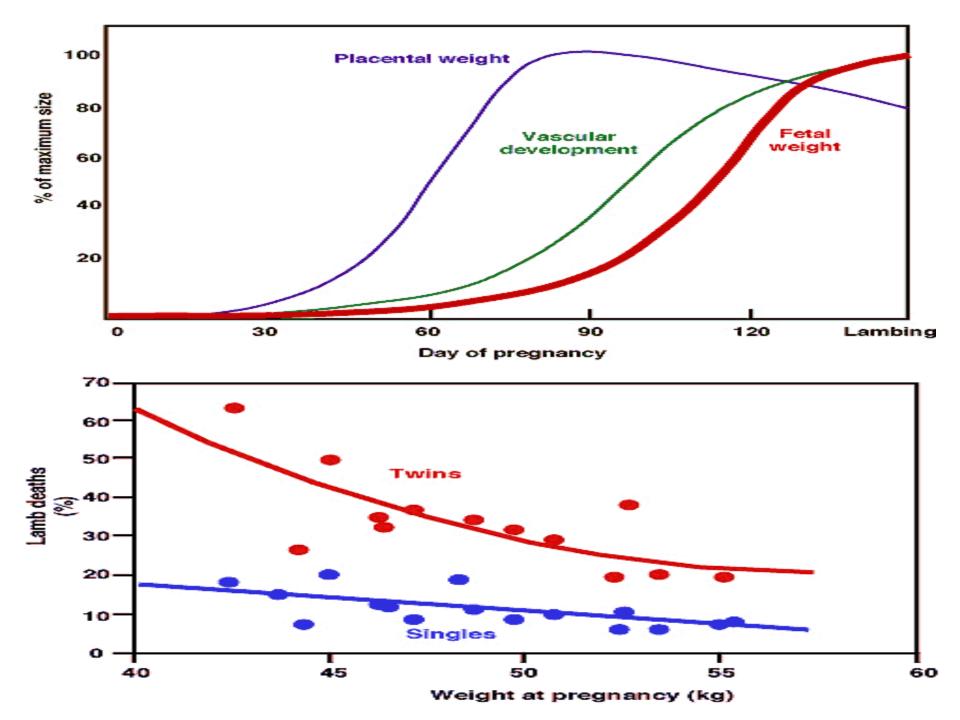
ETIOLOGY

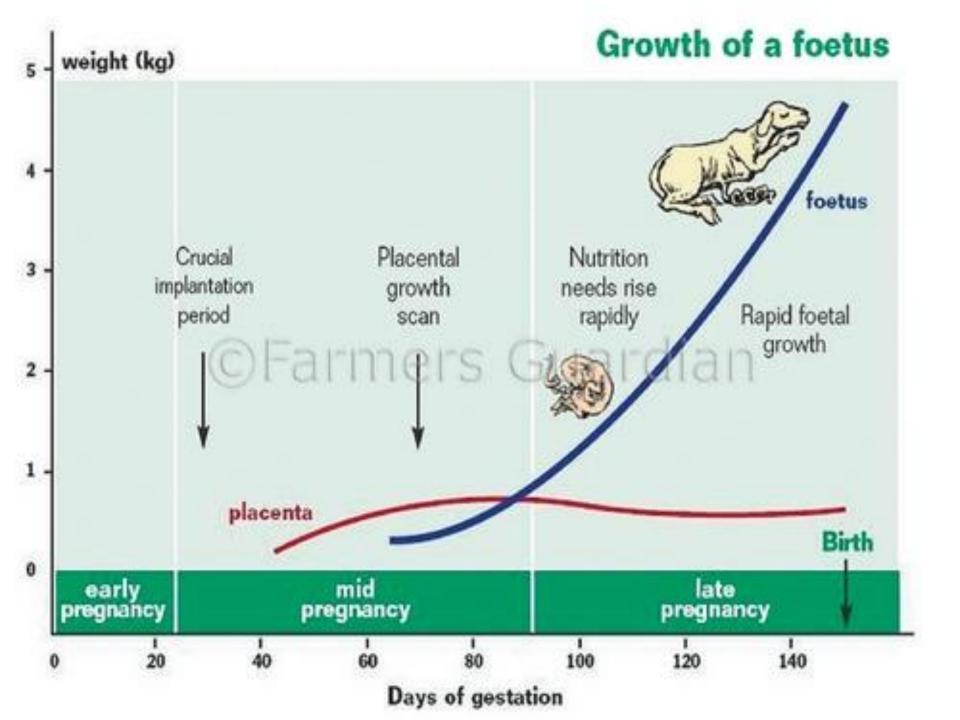
Decline in the plane of nutrition during the last 4 to 6 weeks of pregnancy. This is the period when foetal growth is rapid and the demands for energy markedly increased, particularly in ewes that are carrying twins or triplets.

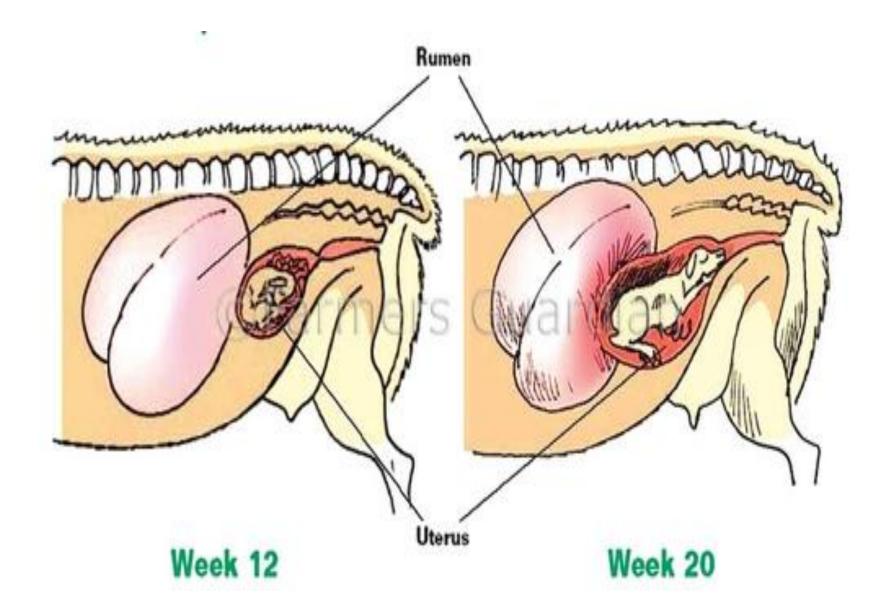
Classification

According to the cause basis to:

- Primary pregnancy toxaemia
- > Fat ewe pregnancy toxaemia
- >Starvation pregnancy toxaemia
- >Secondary pregnancy toxaemia
- >Stress-induced pregnancy toxaemia.







- Primary pregnancy toxaemia
 - fall in the plane of nutrition
- Fat ewe pregnancy toxaemia
- •This occurs without a stress induction. Fat ewes will experience a voluntary fall in food intake in late pregnancy, due to the reduction of the rumen volume by the pressure of intra-abdominal fat and the developing foetus..
- Starvation pregnancy toxaemia
 - •This occurs in ewes that are excessively thin. It is relatively uncommon

Secondary pregnancy toxaemia

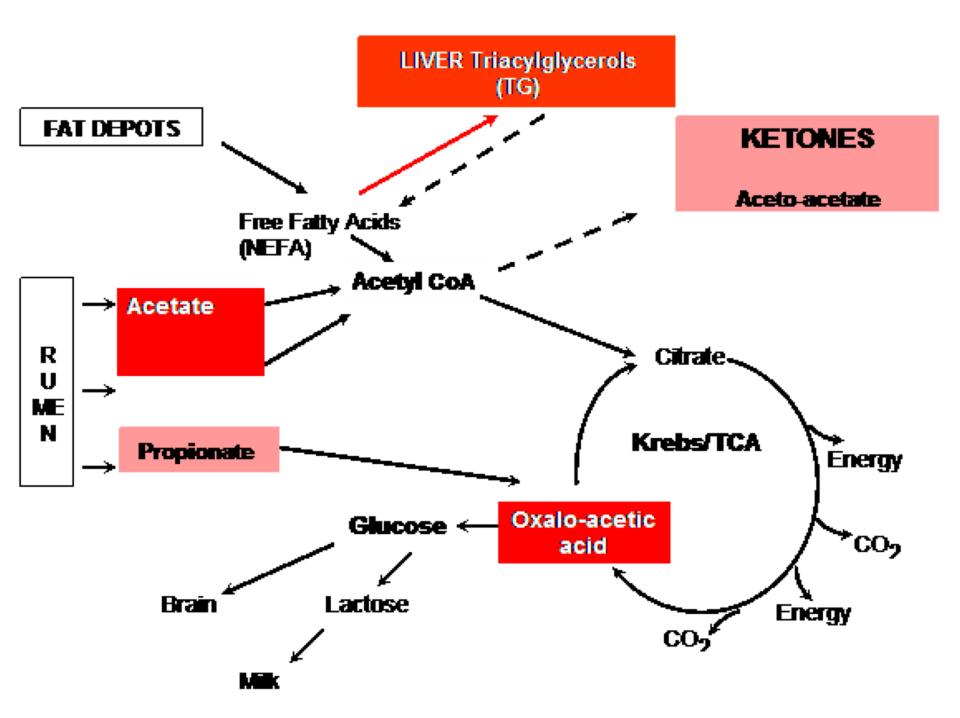
This usually occurs as a sporadic disease as the result of the effect of intercurrent disease such as:

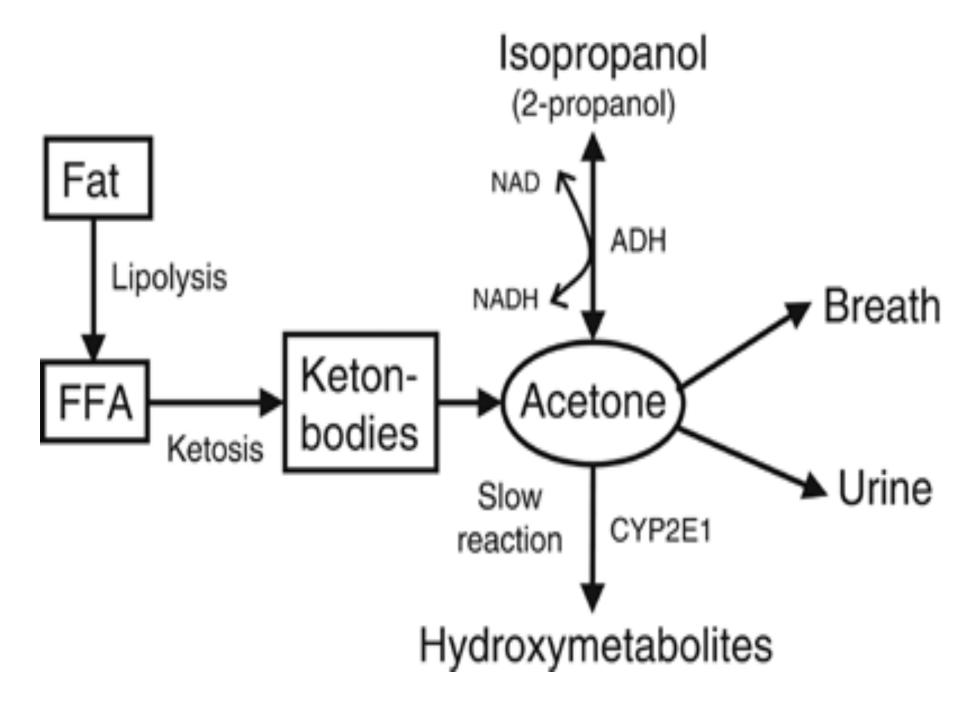
- foot rot or foot abscess, which affects food intake.
- •Heavy worm infestation, e.g. with *Haemonchus contortus*, these cases would add a similar drain on glucose metabolism and increase the chances of development of the disease.

>Stress-induced pregnancy toxaemia

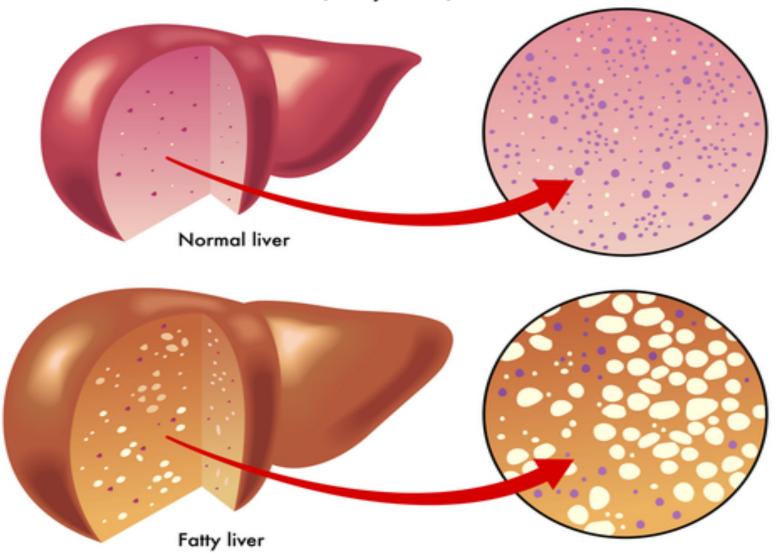
- •This is the least common cause of the disease, one where stress is the initiator.
- •Examples are close shepherding, the transport of late pregnant sheep.

Ewes that are predisposed to the disease have an ineffective gluconeogenic response to the continued, preferential demands for glucose by the growing fetuses resulting in hypoglycemia, lipid mobilization and the accumulation of ketone bodies and cortisol.





Hepatic Steatosis (Fatty liver)



pathogenesis

The subsequent disease and metabolic changes are associated with excessive lipid mobilization.

➤ Elevated concentrations of Beta hydroxybutyrate further suppress endogenous glucose production and exaggerates the development of ketosis and the negative feedback of hyperketonemia on glucose production can result in a vicious circle.

pathogenesis

- The disease manifests with an encephalopathy, hypoglycemic
- encephalopathy ——> hypoglycemia in the early stages of the disease.

•There is an abnormally high level of cortisol in plasma

signs:

- > Weakness, dull attitude, and poor appetite.
- > Separation from the flock or herd.
- **►** Inability to rise.
- >Apparent blindness, staring off into space.
- ➤ Constipation is usual, the faeces are dry and scanty.
- There is grinding of the teeth.









><u>signs:</u>

- ➤ In later stages, marked drowsiness develops and episodes of more severe nervous signs occur. In these episodes, tremors of the muscles of the head cause twitching of the lips, champing of the jaws and salivation.
- The muscle tremor usually spreads to involve the whole body and the ewe falls with tonic-clonic convulsions.

>signs:

- Abnormal postures including unusual positions of the limbs and elevation of the chin the 'stargazing' posture and incoordination and falling when attempting to walk.
- ➤ Recumbent in 3-4 days and remain in a state of profound depression or coma for a further 3-4 days.

signs:

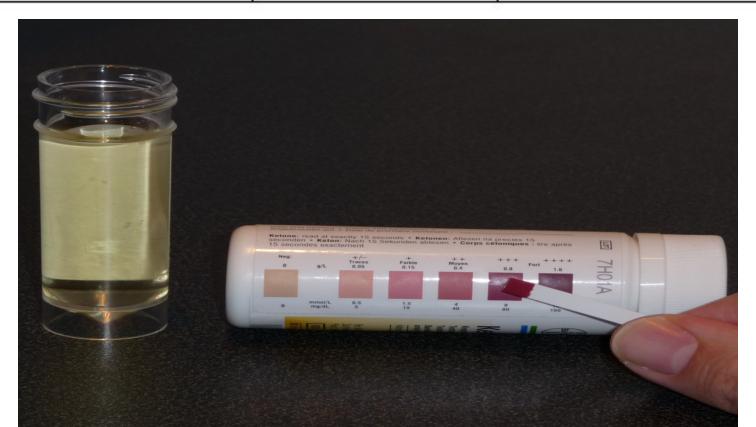
The respirations are rapid, there may be an expiratory grunt

- Foetal death occurs commonly and is followed by transient recovery of the ewe, but the toxaemia caused by the decomposing foetus soon causes a relapse.
- Rumen contractions are weak or absent .

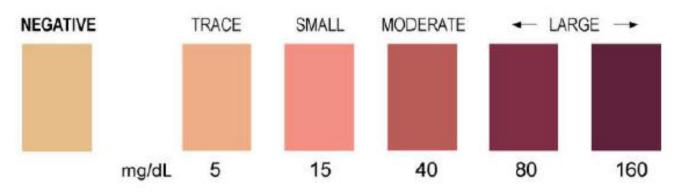
Diagnosis

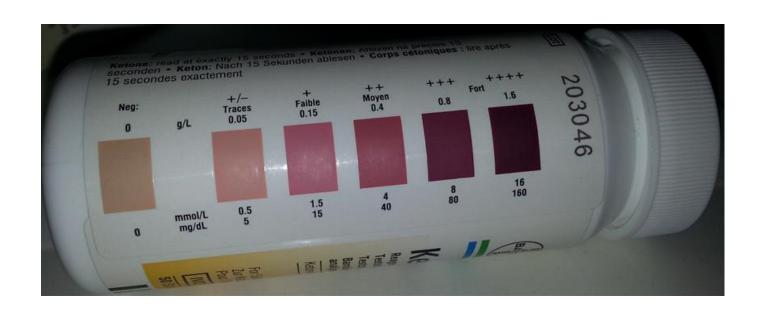
- >History of pregnancy
- >Clinical signs
- >Ketone bodies high in blood and in urine (Acetoacetate 0.24-0.36mg/dl Acetone 0-10mg/dl β-Hydroxybutyrate >5mmol/L)
- ➤ Blood glucose below 25mg/dl.
 - (50 80 mg/dl)

Ketone Body	Normal level	In ketosis
Acetoacetic acid	0.1 mg/100ml	Upto 7 mg/100ml
B-hydroxy butyric acid	8 mg/100ml	Upto 30 mg/100ml
Free fatty acid	9 mg/100ml	Upto 28 mg/100ml



KETONE-Read at exactly 15 seconds.





DIFFERENTIAL DIAGNOSIS

- **≻**Listeriosis
- > Cerebral abscess
- **>**Acidosis
- > Uterine torsion or impending abortion
- **≻**Rabies.

TREATMENT

- Treatment of pregnancy toxemia requires that any predisposing illness, such as pneumonia or foot rot, be adequately treated to eliminate the detrimental effect of that illness on the pregnant mother's appetite
- Sheep treated very early in the course of the disease generally respond favourably, but response to therapy is poor once sheep have become recumbent.



Parentral therapy

➤ Electrolytes and glucose (5% dextrose) given over a prolonged period of time

- ➤ Corticosteroids Isoflupredone/ ISOFLUD {2mg/ml}
- ≻dose;2-5mg)
- >Trenbolone acetate 30mg i/m daily.
- > Triamcinolone

Oral therapy

> propylene glycol or glycerine (110 g/day) given orally is used to support parentral glucose therapy.

Caesarean section

- >Alternate to replacement therapy.
- The demand for glucose by the lambs is immediately removed and both the ewe and the lambs have a high chance of survival providing the caesarean section is conducted before there is irreversible brain damage in the ewe and providing the lambs are close to term. If the ewe is in the recumbent stage then her chance of survival is low.

Prevention:

- ➤ Feed ewes and does appropriately to avoid excessively fat or thin body condition during pregnancy
- ➤ Ultrasound exams at 40-60 days post-breeding can be used to identify ewes and does with twins or triplets, allowing to feed these animals more energy during late gestation.

prevention:

- ➤ Sudden changes in type of feed should be avoided and extra feed provided during bad weather
- > Feed high-quality grass hay and/or alfalfa;
- > Provide plenty of clean, fresh water at all times
- > Parasitic burden should be ruled out