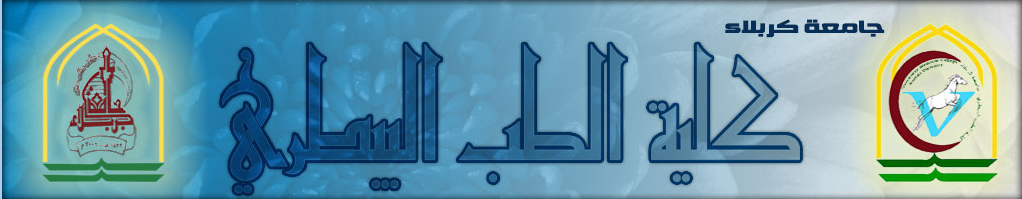
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*Pregnancy Toxemia (Ketosis) in ewes*

*تسمم الحمل في النعاج*

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Pregnancy toxemia in ewes is a disease affecting sheep during late gestation, characterized by feed refusal and neurologic dysfunction progressing to recumbency and death. It is seen more often in older ewes and those carrying multiple fetuses. Pregnancy toxemia is almost never observed in replacement ewe-lambs or yearlings lambing for the first time.(2)

Pregnancy toxemia is a metabolic disorder caused by low glucose concentrations in the blood and excessive breakdown of body fat to compensate. "Ketones" are the toxic by-product produced during this rapid breakdown of fat, and it is possible to test for their presence in the ewe's urine.(2)

**Quick Facts...**

* Pregnancy toxemia in sheep is also known as pregnancy disease, lambing sickness and twin-lamb disease.
* The principal cause of pregnancy toxemia is low blood sugar (glucose).
* Onset of the disease is often triggered by one of several types of stress including nutritional or inclement weather.
* The disease is most prevalent in ewes carrying two or more lambs . The disease also affects ewes that are extremely fat or excessively thin.
* The best preventive measure is increased feeding of high energy concentrates and grains during the last month of pregnancy.(3)

**Occurrence and Causes**

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Pregnancy toxemia in sheep and has also been called ketosis, lambing sickness, pregnancy disease and twin-lamb disease. It occurs in all parts of the world and is an often fatal disease occurring only during the last month of pregnancy. Death occurs in two to 10 days in about 80 percent of the cases. It most often affects ewes pregnant with twins or triplets and is characterized by low blood sugar (glucose). Economic losses because of the disease have been considerable and it is the most commonly occurring metabolic disease of sheep.(3)

**Epidemiology and Pathogenesis**

In late gestation, the liver increases gluconeogenesis to facilitate glucose availability to the fetuses. Each fetus requires 30–40 g of glucose/day in late gestation, which represents a significant percentage of the ewe's glucose production and which is preferentially directed to supporting the fetuses rather than the ewe. Mobilization of fat stores is increased in late gestation as a method of assuring adequate energy in the face of increased demands of the developing fetus(es) and impending lactation. However, in a negative energy balance, this increased mobilization may overwhelm the liver's capacity and result in hepatic lipidosis with subsequent impairment of function. Additionally, twin-bearing ewes appear to have more difficulty producing glucose and clearing ketone bodies, thus increasing their susceptibility to pregnancy toxemia.(3)

***[](http://www.flickr.com/photos/baalands/4205069456/)Symptoms***Does and ewes suffering from pregnancy toxemia (ketosis) appear lethargic, sluggish and often fail to eat. The first symptom noticed in does and ewes is an unwillingness to eat. They become depressed, weak and have poor muscle control and balance later in pregnancy.  
Many times, when they lie down, they are unable to rise. Early in the disease, does or ewes will show a positive test for ketone bodies in the urine. The breath of does and ewes will have a sweet or foul smell. Ketone bodies are by-products of fat breakdown found in the blood and urine. Test kits are often available for ketone bodies hypocalcemia (or milk fever) Ewes in early stages of pregnancy toxemia will go off feed and appear lethargic. Their heads droop and they lag behind the rest of the flock and walk aimlessly. Teeth grinding and twitching is common. Eventually, affected ewes become depressed, weak and have poor muscle control. In latter stages, they lie down and are unable to rise. If left untreated, coma and death result(4)

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Postmortem changes demonstrate varying degrees of fatty liver, enlarged adrenal glands, and often include multiple fetuses in a state of decomposition indicating premortem death. Very thin ewes may appear starved (eg, serous atrophy of the kidney and heart fat). However, these signs alone are not pathognomonic for death due to pregnancy toxemia. Postmortem samples of aqueous humor or CSF can be analyzed for β-hydroxybutyrate (BHB). Levels >2.5 and 0.5 mmol/L, respectively, are consistent with a diagnosis of pregnancy toxemia.(2)

. ***Transmission***Pregnancy toxemia is not a transmittable disease from one doe or ewe to another, however, flocks or herds experiencing the disease may appear to be infectious because the plane of nutrition and management throughout pregnancy is similar. This shortage of energy in the diet may cause these symptoms in several does or ewes, especially those carrying twins or triplets.(2)

**Diagnosis**

Laboratory findings in individual ewes may include hypoglycemia (often <2 mmol/L), elevated urine ketone levels (evaluated by commercial qualitative test tablets), elevated BHB levels (normal <0.8 mmol/L, subclinical ketosis >0.8 mmol/L, and clinical disease >3.0 mmol/L), and occasionally hypocalcemia. Hypoglycemia is not a consistent finding, with up to 40% of cases having normal glucose levels and up to 20% having hyperglycemia. If the diagnosis needs further confirmation, CSF glucose levels may be more accurate than blood; they remain low even when serum glucose rebounds in advanced cases after fetal death. BHB is a more reliable indicator of disease severity than are blood glucose levels. Nonesterified fatty acids can also be elevated above 0.4 mmol/L, indicating likely hepatic lipidosis resulting in impaired hepatic (2)

**Treatment**

Treatment of advanced cases of pregnancy toxemia is often unsuccessful. Propylene glycol can be given orally that acts as a precursor to glucose. The addition of calciumm, potassium and insulin is also useful. Hypoglycaemia can also be treated by administering IV dextrose, followed by an oral electrolyte solution.

If biochemistry reveals a hypocalcemia, this can be corrected by giving calcium IV. Severe ketoacidosis can be treated by administering oral potassium chloride (KCl). The contributing factors (eg, nutrition, housing, other stressors) should be corrected for the group and feeding management assessed (eg. adequate feeder space, feeding frequency, protection from adverse weather).

A blood sample should be taken from a number of ewes in late gestation and glucose and BHB levels measured. This will determine if there are any problems in the rest of the flock.

If it is thought that the ewe has aborted then prophylactic [antibiotics](http://en.wikivet.net/Antibiotics) should be administered. If foetuses are alive a caesarian section should be considered.(1)

**Prevention**

Ewes should be in a good body score at tupping and should maintain a good body condition score throughout gestation. Good body condition can be maintained by good management of feeding. Ewes should receive grain as a carbohydrate source and also a protein source so that the [rumen](http://en.wikivet.net/Rumen_-_Anatomy_%26_Physiology) microbes can make use of the carbohydrate. It is also important to separate out thin sheep and feed them away from the main flock. Ideally, ewes should be ultrasound scanned to determine the number of foetuses they are carrying and then separated into groups and fed accordingly.(1)

**References**

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4 [-Pregnancy toxemia (ketosis) in does and ewes - University of Missouri](http://www.case-agworld.com/cAw.LU.ket.html)  
[Pregnancy Disorders (Metabolic) in Transition Ewes - Ontario, Canada](http://www.omafra.gov.on.ca/english/livestock/sheep/facts/03-017.htm)  
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[Ketosis or Pregnancy Toxemia in the Doe - SweetLix Livestock Supplement System](http://www.sweetlix.com/media/documents/articles/Goat_Ketosis%20or%20Pregnancy%20Toxemia%20in%20the%20Doe.pdf)