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KOONAC Goat Farm

Pregnancy Toxaemia in Goats

1. Introduction

Pregnancy toxaemia in goats is normally seen as a rather uncommon disease[1], and the risk this disease can pose to goats, especially if they are healthy and well-fed, is therefore often underestimated.

The survival rate for goats with pregnancy toxaemia is generally expected to be low. In a commercial goat dairy in Portugal with 1700 goats, for example, 86% of the affected goats died within 14 to 58 hours once clinical signs of pregnancy toxaemia were observed.

Early symptoms of pregnancy toxaemia might be vague and unspecific, but they exist and usually occur much earlier than only a few days before the goat dies. Since pregnancy toxaemia can be fatal if not early and correctly diagnosed, early identification of goats at risk and of those symptoms that foreshadow an upcoming pregnancy toxaemia are the keys to avoid fatalities and to increase the chances of survival and recovery.

The goal of this text is to describe this disease and raise awareness for its early symptoms, and also to describe measures that can be taken to successfully manage such critical situations.

2. Cause

Pregnancy toxaemia is a metabolic disorder. It occurs in the last weeks of pregnancy, and is caused by insufficient energy intake of the doe to satisfy its energy requirement, resulting in a negative energy balance. 80% of foetal growth takes place during the last 40 days of pregnancy, and during this time the embryos drain more and more glucose, which is their principal source of energy, from the blood of their mother. Glucose requirements during late pregnancy are more than twice as high as in non-pregnant does (Figure 1), and the blood sugar level of the pregnant doe drops to about half of the non-pregnant doe, from 35-45 mg glucose per 100ml of blood to 20-25 mg/100 ml. Pregnancy toxaemia may develop when the level drops below about 18 mg/100ml.

[1] McGregor, B. A.: Nutrition of goats during drought. RIRDC, March 2003.

It is interesting to note that the foetus maintains a much lower plasma glucose concentration[2] than its mother, which enables the placenta to still transfer glucose from the mother's blood to the foetus, even if the glucose concentration in the maternal blood is very low.

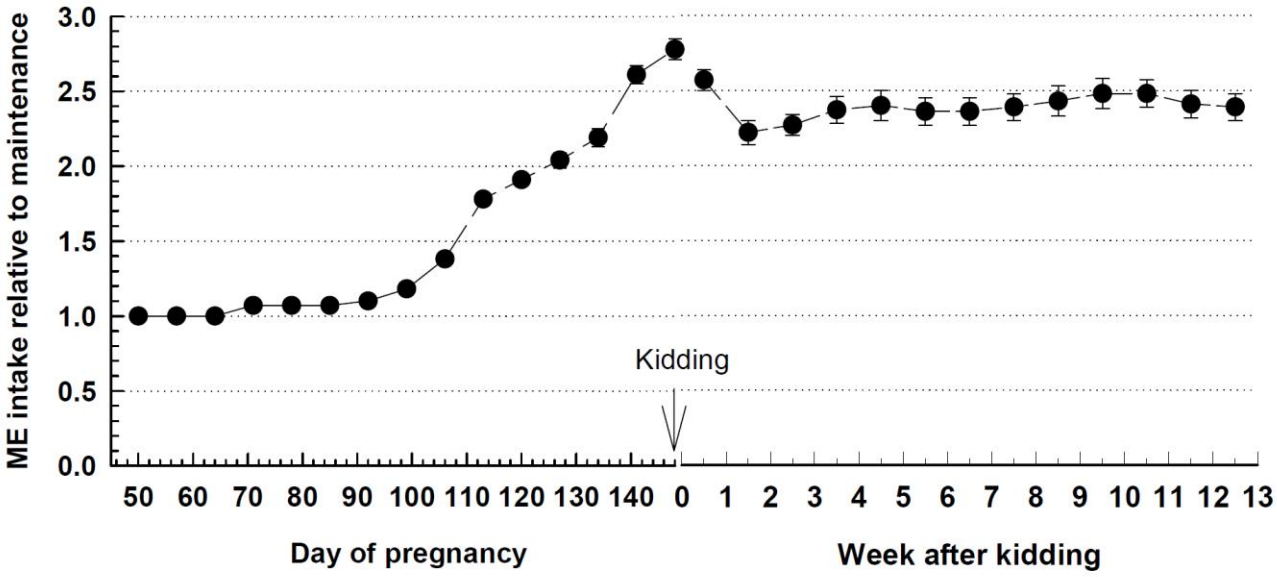


Figure 1: Required metabolisable energy (ME) relative to maintenance ME requirements before and after kidding (from B. A. McGregor, Nutrition of goats during drought, RIRDC, March 2003).

The energy intake of the doe depends on the availability and quality of feed, and on the fitness of her digestive system. However, not even under optimal conditions will the goat be able to eat and process enough feed to match the energy requirements during these critical weeks just before and after kidding. It is, therefore, increasingly forced to mobilise energy from her own body (fat deposits, muscle tissue). These alternative energy resources are metabolised to glucose by the liver[3]. However, the capacity of the liver to break down fatty acids and amino acids is limited, and if its capacity is overwhelmed, the fat will be deposited in the liver instead of being metabolised, resulting in hepatic lipidosis (fatty liver), and ultimately an impaired liver function. Furthermore, it will produce toxic ketones that are released into the blood (Figure 2). As ketosis increases, the bicarbonate level in the blood decreases, resulting in acidosis.

[2] Glucose concentrations in sheep embryos is around 8mg/100ml; no comparable data could be found for goat embryos.
[3] Fatty acids and glycerol from the goat's body reserves are oxidized to form Acetyl-CoA (coenzyme A). The liver cannot cope with high levels of Acetyl-CoA, and if the liver's capacity is exhausted, some of the Acetyl-CoA is converted into ketone bodies (including Beta-hydroxylbuterate, BHBA)

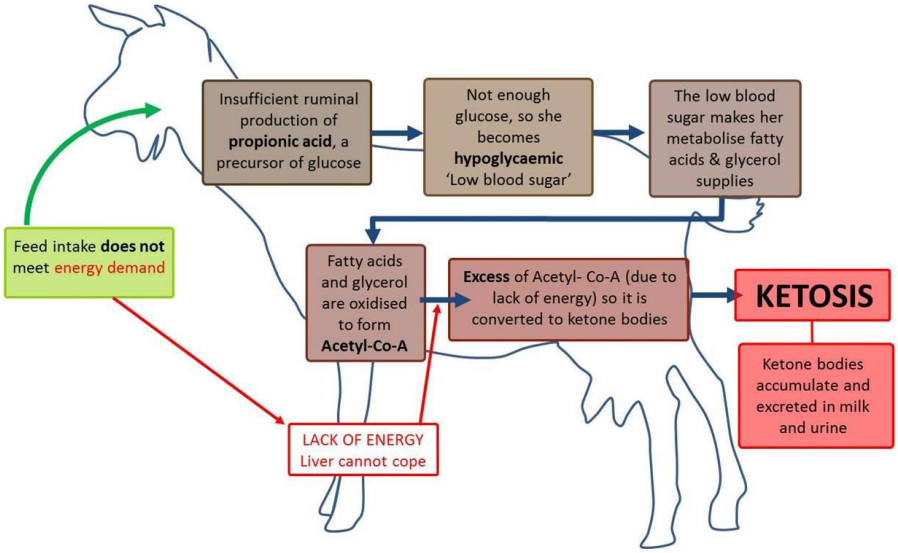


Figure 2: Negative energy balance in pregnant or lactating goats that ultimately can lead to ketosis (from Farm Health Online).

A similar imbalance between energy intake and requirement, leading to similar symptoms, can also occur during peak lactation, usually 2-4 weeks after kidding. This postparturient toxaemia is commonly called "Ketosis" or "Fatty Liver Syndrome".

3. Predisposing factors

There are many factors that can lead to pregnancy toxaemia. Animals where several of these adverse factors apply together are at highest risk, for example older, overfed goats with multiple foetuses, or undernourished animals with an infection (e.g. foot abscess).

3.1. Litter size (multiple foetuses)

The amount of glucose required is directly related to the number of foetuses a doe carries. At our farm, the average litter size is 1.94 kids per birth, of which 19.0 % were single births, 66.4 % were twin births, 13.3 % were triplet births, and 1.3 % were quadruplet births. With multiple births, in particular with triplets and quadruplets, the rumen volume is compressed by the foetuses, which decreases voluntary food intake. Triplets or quadruplets kids at birth can weight more than 20% of the does dry weight (Table 1). The energy requirement of these foetuses exceeds the limits of the goat's energy metabolism, which likely will can result in pregnancy toxaemia, if no corrective measurements are taken.

In the above-mentioned large goat dairy in Portugal, litter size had the highest statistical correlation to pregnancy toxaemia ($p < 0.001$) of all parameters that were included in the study. On

average, those does that were affected by pregnancy toxaemia had 2.7 kids, compared to 2.0 kids per doe in the control group.

Table 1: Cumulated birth weights of all kids from one litter, and percentage of the mother's dry weight (usually at joining) for 632 births at our farm.

	Average	Minimum	Maximum
Singles (n=120)	4.20 kg (7.96%)	1.73 kg (4.03%)	6.15 lg (12.38%
Twins (n= 419)	7.77 kg (13.54%	3.30 kg (5.48%)	11.5 kg (23.24%)
Triplets (n=84)	10.72 kg (15.54%)	6.23 kg (8.71%)	15.70 kg (21.69%
Quadruplets (n=8)	13.19 kg (16.47%)	7.82 kg (14.26%)	18.58 kg (23.97%)

3.2. Obesity

In overfed goats with insufficient exercise fat deposits around the rumen reduce the rumen capacity, which decreases food intake. The energy metabolism of such a goat is not fit, and hence not capable of adequately respond to a substantially increased energy demand. As a result, excessive quantities of (readily available) body deposits are mobilised and deposited in the liver, leading to fatty infiltration of the liver and hepatic dysfunction.

3.3. Undernutrition

Undernourished goats in poor condition have little to no energy reserves they can mobilise to satisfy the increased energy demand during the last weeks of pregnancy. Undernutrition, as a consequence of inadequate food supply, occurs mainly in extensively kept herds of (meat) goats, when goats are held at high stocking density on poor pasture. A heavy worm burden and/or unbalanced rations, which often accompanies inadequate food supply, will further aggravate the problem.

We experienced this situation of severe undernutrition once with a group of goats we rescued from another farm. All animals of this herd were in an awful condition. They all were heavily infected with worms (dominantly Barber's pole worms), and anaemic. One goat that was heavily pregnant was only weighing 42.2 kg (her "normal" body weight later was around 70 kg). She was very weak and could not get up on her own. We gave her 2 x 50 g of glucose (1:5 diluted with water) daily for some days, and also some PRONTO against her scour (containing 85% dextrose mono-hydrate and sodium chloride, sodium bicarbonate, and vitamin C). Soon she was walking around again and was eating ok. Obviously, the sugar boost we had given her had raised her blood-sugar level to a

sufficient value. 10 days later she had twins. Both kids were very small (1.90 kg, and 2.04 kg, respectively). Only one kid survived.

So called “shy feeders” are also at risk of undernutrition, because more dominant and more aggressive animals prevent them from having adequate access to otherwise abundant feed (“round bale feeder syndrome”).

3.4. Stress factors

Basically, every stress factor that affects the animal’s ability to eat enough during late pregnancy can lead to pregnancy toxaemia. Such factors are

- Fear
- Sudden change in feed
- Severe weather
- Poor housing
- Infections (e.g. hoof abscess)
- Transport or dislocation to an unknown environment

3.5. Age

Pregnancy toxaemia more often occurs in older, fully grown goats than in young first-kidders. This is probably because young goats are still growing, and their metabolism is therefore fitter than in older animals. Furthermore, young animals normally have fewer fat deposits, which can prevent the digestive system from adequately “gear up” to meet the increasing energy demand during late pregnancy or during the first weeks of lactation, as it is more likely to occur in older goats.

3.6. Genetics

How well a pregnant goat with multiple foetuses can handle the increased energy demand varies, depending on the genetic predisposition of an individual goat. No scientific reports could be found that would quantify the heritability of this particular aspect of the metabolism. However, based on our own observation we suspect that a predisposition for pregnancy toxaemia can be passed down from the mother to her daughters. Hence, some gene lines in a herd might be more at risk to develop pregnancy toxaemia than other ones.

4. Symptoms of Pregnancy Toxaemia

Below is a list of clinical symptoms that may occur with pregnancy toxaemia. However, it is important to understand that these symptoms may or may not show up, and that symptoms are often vague and non-specific (i.e. also be caused by other problems) at the onset of the disease:

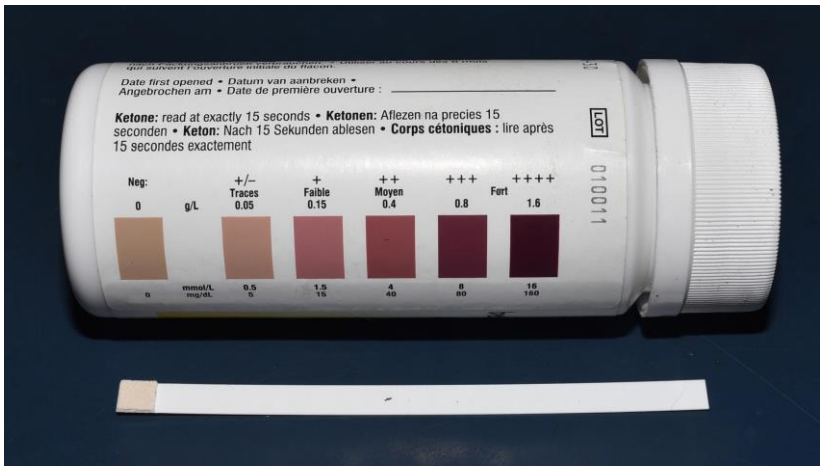
4.1. Inappetence

Initially, the goats are still eating prunings and hay, but increasingly refuse to eat concentrates. Later, they stop eating altogether (anorexia).

Unwillingness to eat concentrate, but still having good appetite for browse and hay is an early warning symptom of an upcoming pregnancy toxaemia. Goats that show this symptom need to be closely observed. In particular, they should be tested for ketones.

The production of ketone bodies is a reliable early indication of an insufficient energy intake that forces the goat to metabolise energy from her body reserves (fat deposits and muscle tissue, see Figure 2), which may result in pregnancy toxaemia.

A cheap and easy do-it-yourself method to measure ketones is using “Ketostix”. These are test strips for humans to self-assess the ketone content of their urine, in particular if they suffer from diabetes or are on a low-energy diet. They are available from every pharmacy. Because the metabolic reaction to a glucose deficiency in humans is the same as in goats, “Ketostix” can also be used to determine the ketone content of goat urine.



Picture 1: “Ketostix” are used to determine the ketone content of urine.

Whereas the urine of healthy goats does not contain ketones, the concentration of affected goats can exceed 1g/l. In this case the test-pad of the “Ketostix” turns to a dark-violet (see Picture 1).

4.2. “Squinty eyes”

One goat breeder had observed that “squinty eyes” can be an early sign of low blood sugar. When the blood sugar of the goats drops, they get a headache, and they squinch their eyes. This is also accompanied by the sweet smell of their breath.

4.3. Weight loss

Several of our goats, which had triples or quadruplets and developed pregnancy toxaemia, lost condition in the last weeks before kidding and got very skinny.

One such case was “Louise” (see Picture 2). She became very skinny towards the end of pregnancy, but otherwise did not show the obvious symptoms of pregnancy toxaemia. However, after parturition her condition did not recover. Her appetite remained poor, and she stayed emaciated. We suspect that vital organs had been irreversibly damaged during the last weeks of pregnancy, and that these damages prevented her from recovering to her previous condition.

Picture 2: “Louise” with her four kids, 5 days after kidding

4.4. Unwillingness to move

Goat walks with difficulty, legs may swell. Later recumbency.

Pain in the joints is a symptom of a fairly advanced pregnancy toxaemia. It often comes together with abscesses and/or granuloma in the hoofs. It is important to understand that the unwillingness to walk is not only a result of these hoof problems, but is also directly caused by pregnancy toxaemia.

4.5. Other symptoms of advanced pregnancy toxaemia

- Separation from the herd, mild depression.
- Occasionally the goat displays some nervous signs (tremor around the head and ears, reduced vision or even blindness, stargazing);
- In the final stage coma, with or without abortion, and death.

5. Steps to avoid pregnancy toxaemia

5.1. Identify risk candidates

All pregnant goats should be assessed for their risk to develop pregnancy toxaemia, based on the predisposing factors listed in chapter 3, and the risk candidates need to be closely observed throughout the end of the pregnancy.

Older goats, which are more on the “well-fed” site, have to be considered as risk candidates. This is particularly the case if they are having triplets or even quadruplets.

With some experience, litter size can be roughly guessed: We found that goats with 3 or 4 embryos often dry off earlier in pregnancy than those with only 1 or 2 embryos, and also that their udder grows earlier and bigger. However, to exactly know the litter size, an ultrasound by a veterinarian is required.

Another strategy to reduce the risk for pregnancy toxaemia is to daily supplement all pregnant goat with a small amount of molasses.

5.2. React to early symptoms

As soon as a goat, in particular one of the risk candidates, shows some of the symptoms listed in chapter 4, it should be tested for ketones. This can be done by either have a blood sample of the goat analysed by a veterinarian, or by using “Ketostix” (see chapter 4.1).

5.3. Treatment

Because the prime reason for enterotoxaemia is a glucose deficiency in the blood of the mother, supplementing the pregnant goat with additional glucose or dextrose is the easiest way to correct this glucose-imbalance. This can be done by either drenching or intravenously injection of 100 to 200 ml of a 50% dextrose of glucose solution. Alternatively, 120 to 180 ml of glycerine of propylene glycol, mixed with an equal volume of water, can be orally given 2 to 3 times daily. To help restore glucose uptake, of 20 to 40 IU protamine zinc insulin can be administered intramuscularly every other day.

One good example for a successful treatment was “Nelly”. Her expected date of kidding (EDOK) was 18. June 2022. During the first weeks of May 2022 (about 3-4 weeks before EDOK), “Nelly” increasingly refused to eat her pellets, but her ketone readings on 16th and 24th of May 2022 were very low (<0.05 g/l). Similar low values of ketones were also found in the urine of other pregnant goats which we had identified as potential candidates for pregnancy toxaemia.

However, whereas the readings of all other goats remained low, the reading for Nelly’s urine jumped up to 0.8 g/l on 5. June (two weeks before due date). This was an alarmingly high value, indicating that Nelly had a substantial energy intake deficiency and was forced to mobilise energy from her own body fat. To support her energy (glucose) requirement, we immediately started to

give her 50 g glucose (diluted in 250ml of warm water) twice daily. On 7. June. after two days of this treatment, the ketone reading had dropped back to 0.15 g/l. We continued to supply “Nelly” with 50 g of glucose once per day until 15. July, when she successfully gave birth to triplets.

During these last days of pregnancy, “Nelly” never showed any of the typical clinical symptoms for pregnancy toxaemia.

Picture 3: Nelly (picture taken 30. May 2022)