

Pregnancy Toxemia
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Pregnancy toxemia is one of the issues that breeders and fanciers must deal with in pregnant sows. Unfortunately it is a syndrome in which treatment is often unrewarding, however there are some sows that can be saved. There are actually two forms of toxemia that are seen and they are sometimes impossible to differentiate clinically.

The more common form that is seen and identified as pregnancy toxemia (AKA pregnancy ketosis) is a metabolic disorder affecting the energy balance in the sow. This form is similar to what is observed in heavily pregnant sheep where they are in a negative energy situation due to the drain of multiple and/or large fetuses. The sow cannot keep up with the energy demands of the unborn litter. These sows become ketotic. (Ketones are a product that builds up in the blood when fat or fatty acids are used as a fuel source.) As the sows become lethargic and unwilling to eat, they may progress rapidly towards death. Other signs that may be seen in affected animals are difficulty or rapid breathing (this may be due to compression of the chest cavity by a distended abdomen from the large fetal load- or may be a primary sign related to the ketoacidosis). The sows may appear uncoordinated and may even suffer muscular spasms. The animals seen with this form of pregnancy toxemia are generally those sows who are overweight, have a large litter size/large gravid uterus, little space to exercise and other sorts of dietary or environmental stresses.

The signs may start several weeks before the expected parturition (birthing) date.

Treatment for this form of pregnancy toxemia consists of reversing the ketosis and metabolic imbalance by increasing caloric intake. This can be done via IV fluids, but more often by subcutaneous fluid administration in which 50% dextrose has been added (dilute to a greater degree with SQ fluids). Oral glucose can be given and the sow started on forced feedings. Almost any item containing calories that is acceptable to the pig can be used for forced feedings: KMR, strained baby carrots, pellet fines, etc. Commercial preparations such as Oxbow's Critical Care are excellent for these animals. Some also recommend administration of calcium gluconate especially in the face of animals with muscle spasms or those who are "flat out". Feed small amounts often remembering that gastric emptying time in cavies is two hours. Resolution of the signs generally disappear within a few days after the litter is born and the sow's appetite returns. Continued monitoring is warranted however as the demands of the pups increase.

(Anecdotally, the sows that I have managed to save from this form of toxemia and have rebred uniformly have suffered more severe manifestations with each subsequent pregnancy, to the point that once an animal has ketosis in my herd, that animal is removed from the breeding population. There has been damage done to the liver and kidneys which I feel is simply compounded by subsequent insults of additional pregnancies. These animals are much harder to pull through subsequent pregnancies. In my mind the conditions that first led to toxemia are likely still present in those sows. This is in

contradistinction from sources who state that this form of pregnancy toxemia is more prevalent in first or second pregnancies – maybe because they don't live for the 3rd or 4th! This is however just my opinion. I do not see it in just the first or second pregnancies as suggested by some (Suckow), but an additional risk factor in my herd would include increased age- that is however just my experience in my herd.)

The second manifestation of pregnancy toxemia seen in covies is more akin to preeclampsia seen in women and is felt to be a circulatory issue. During late pregnancy the renal arteries may be compressed resulting in hypertension, or conversely the animal may be suffering low blood pressure as the disease progresses into shock. Additionally the gravid uterus compresses the aorta posterior to the renal arteries which results from decreased blood flow to the uterus. This may lead to infarction (area of necrosis due to lack of circulation) of the placentas and may lead to DIC (disseminated intravascular coagulation). These animals may die with very little warning- the sow looks uncomfortable- perhaps a bit off- and then boom, she is dead. Treatment of choice is emergency cesarean section and supportive therapy with fluids in the case of shock. In humans, magnesium sulfate is used to treat preeclampsia and has been variably successful with inconclusive results in guinea pigs, so that this is not really something offered in standard care for treatment of the circulatory form of pregnancy toxemia.

When facing pregnancy toxemia it is important to note that prevention is a much better approach than trying to treat the sick sow. Sows should probably be carrying less condition than wanted for a show animal; they should have feed and fresh water available at all times. Pregnancy is not the time for changing housing, roommates, feed or any sort of nutritional or environmental condition. A pregnant sow that doesn't eat should be viewed with great concern and action taken sooner than later. You will be able to save some, you will lose many more. However, predicting which ones will respond to therapy and which ones will not remains elusive.

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