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Pregnancy Toxemia in the Ewe

Leanne M. Schulz*
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INTRODUCTION

Pregnancy toxemia, also known as ovine ketosis, lambing sickness, twin lamb disease, or domzietke, is a widespread, usually fatal disease of sheep.1,2,3,4,5 The disease also occurs occasionally in goats, and the clinical course is very similar to that in sheep.3,5

Pregnancy toxemia occurs during late pregnancy, and although death losses in a given flock are usually sporadic, they can exceed 13%.1 Among severely affected ewes, the death rate often approaches 95%.1 These factors, combined with the prevalence of the disease, make pregnancy toxemia of major economic importance to the sheep industry.

Pregnancy toxemia is significantly different from bovine ketosis, even though ketosis is a major factor in both diseases and the clinical signs are often similar.7,8 The two major differences are the time of onset and the response to treatment.9,10 Pregnancy toxemia occurs prior to lambing, while bovine ketosis occurs from a few days to several weeks post-calving. Pregnancy toxemia does not respond readily to any treatment, unlike bovine ketosis, which responds quickly to intravenous glucose combined with the administration of either propylene glycol orally or glucocorticoids intramuscularly.

INCIDENCE AND PREVALENCE

Although most common in ewes carrying at least two fetuses, pregnancy toxemia can be seen in ewes carrying a single large fetus.1,6,7 Published literature indicates that the disease is seen during the second and subsequent pregnancies, but the authors believe that it may also occur in ewe lambs during their first pregnancy. Elderly ewes often have a higher incidence because poor teeth, parasites, disease, and other age-related syndromes affect their plane of nutrition and therefore their body condition.2

The disease occurs during late pregnancy and is nearly always seen in ewes that are either very thin or are overweight at this time.4,5,6,7,11 This indicates that a ewe in moderate flesh during late gestation is the ideal type not to have the disease. Also, the disease is more common in pasture and farm flocks than in range flocks, possibly because range flocks receive more exercise.4,7 Pregnancy toxemia occurs almost exclusively in the spring, but only because nearly all ewes are bred to lamb in the spring.4

Pregnancy toxemia occurs in all areas of the world in which sheep are raised.4,5,11 The incidence has been reported to be higher than average in areas of New Zealand, Australia, Britain, South Africa and the United States.4 This may be due to differences in management of flocks in these areas, but more likely it is because these are the major sheep producing areas of the world and thus the disease is more likely to be recognized and reported in these areas.

PATHOGENESIS

Most authors are in agreement that pregnancy toxemia is caused primarily by a reduced food intake during late gestation.1,4,5,6,7,11 This reduced intake may occur in both thin and fat ewes that are being malnourished. However, in fat ewes it may occur as a result of inadequate capacity of the stomachs due to compression by body fat in combination with a large, gravid uterus.6 It may also occur in both thin and fat ewes that voluntarily reduce their food intake due to some stress such as worming, shearing,
Seventy percent of fetal growth occurs during the last six weeks of pregnancy, and this growth causes a large demand for glucose by fetal tissues. If the ewe’s energy intake is insufficient to supply the fetal demand plus her own needs for glucose, the ewe’s tissue stores of glucose are drawn upon to meet these needs. When these tissue stores are used up, hypoglycemia occurs, which in itself causes some central nervous system depression. Also, when there is a hypoglycemic state present, fatty acids begin to be mobilized from fat stores in the body in order to meet the body’s energy demands. These fatty acids are oxidized in the liver, which produces acetyl-CoA as one of the metabolites. Normally, acetyl-CoA is utilized in the tricarboxylic acid cycle and forms nontoxic end-products. However, when there is a deficiency of glucose in the body, this acetyl-CoA cannot be utilized in the tricarboxylic acid cycle and is instead used in a biochemical process that produces ketone bodies as its end-product. The resulting ketosis leads to an acidosis, and this ketoacidosis causes an increase in severity of clinical signs and eventually contributes to the death of the ewe.

CLINICAL DESCRIPTION

There are two major syndromes of the disease: the malnutrition syndrome and the stress syndrome. In the malnutrition syndrome, the disease is produced because of an ongoing lack of glucose precursors in the diet, which results in the above events taking place. In the stress syndrome, the ewes are maintaining a normal glucose metabolism until some stress such as sorting or shearing causes them to go off feed, which reduces the availability of glucose precursors from the diet and shifts the ewe’s metabolism to the production of ketone bodies. Clinical signs are subtle in the early stages and may go unnoticed. Initially, affected ewes merely seem sluggish and often lag behind the flock when it moves. Signs gradually progress over the next 2–10 days to include anorexia, incoordination and weakness. In the late stages, severe neurological signs such as blindness, muscle tremors, convulsions and coma progress and eventually cause the death of the ewe.

In the early stages of the disease, clinical pathology shows a hypoglycemia, increased plasma levels of fatty acids and glycerol, and hyperketonemia. There may also be ketonuria, and plasma cortisol levels are elevated in an attempt to increase blood glucose levels. (One author has suggested that this increase in plasma cortisol may be the precipitating cause of pregnancy toxemia rather than being a secondary change, but this theory is not widely accepted.) In advanced cases showing acidosis and concomitant renal failure, plasma bicarbonate levels will be low and blood urea nitrogen levels will be elevated. In terminal stages, there may be a shift to a hyperglycemia in response to the elevated plasma cortisol, but by this stage the central nervous system has become unresponsive to glucose and death is usually inevitable.

NECROPSY

The following lesions, when found at necropsy, are strongly indicative of pregnancy toxemia. The uterus almost always contains two or more fetuses, although in rare cases a single large fetus may be present. These fetuses may be found in various states of decomposition if fetal death occurred prior to maternal death. The liver is pale, enlarged and friable due to fatty infiltration which occurs as the body mobilizes large quantities of fat depots. An analysis of fat content of the liver may show a rise from the normal 3% to a value exceeding 30%. In some instances, they may be greater than 65% larger than normal. This enlargement of the gland is a secondary hypertrophy that occurs as the gland is stimulated to produce more cortisol in an attempt to raise blood glucose levels. Grossly, the adrenal cortex is dark in color, while the medulla is lighter than normal. Other lesions that are inconsistently seen include pale, fatty kidneys and heart, and distended mesenteric blood vessels.

None of the above lesions by itself is pathognomonic for pregnancy toxemia. However, when seen in combinations of several or all of the above, these findings are highly suggestive of pregnancy toxemia, especially if the lesions are seen in a carcass that is either emaciated or obese.

DIAGNOSIS

Pregnancy toxemia is unique in that the symptoms are primarily central nervous system in origin, and that these symptoms develop on-
ly during the last 3–6 weeks of gestation. Also, these CNS signs are accompanied by fatty liver, hypoglycemia and ketonuria.\(^4\)\(^1\)\(^1\) When confronted by these typical clinical signs and laboratory findings as described above in combination with the typical post-mortem lesions, a diagnosis of pregnancy toxemia is strongly indicated.

The major differential diagnosis that must be considered is hypocalcemia.\(^4\)\(^6\) Both diseases show CNS signs; however, these signs are usually more pronounced and varied with pregnancy toxemia. In hypocalcemia, nervous signs are usually limited to tremors and dullness which progress to coma and death.\(^6\) The course of the disease is usually much shorter with hypocalcemia, death occurring within 24 hours in untreated cases. In contrast, death may not occur until 5-7 days after the first appearance of clinical signs in cases of pregnancy toxemia.\(^4\) Also, with hypocalcemia there is a rapid and dramatic response to calcium salts administered intravenously. This treatment is not beneficial in cases of pregnancy toxemia.\(^4\)\(^7\)

Other sporadically occurring diseases that may produce nervous signs and occasionally be confused with pregnancy toxemia include listeriosis, rabies, cerebral abscesses, otitis media, infestation with Coenurosis cerebralis larvae, and loping ill. However, these diseases can usually be differentiated by such factors as duration and severity of clinical signs and morbidity rates in the flock. Also, many of these diseases show characteristic gross and histological post-mortem lesions and thus should not normally be mistaken for pregnancy toxemia.\(^7\)\(^12\)

**THERAPY**

In general, treatment of pregnancy toxemia is only moderately successful even if started in the early stages of the disease. If treatment is not initiated until the ewe is comatose, the mortality rate approaches 100% in spite of intensive therapy.\(^4\)\(^6\)\(^7\)\(^16\)

One of the earliest treatments for pregnancy toxemia consisted of either oral or intravenous administration of glucose.\(^4\) Although this seems to be a very logical treatment when faced with hypoglycemia, results were not promising. In some trial studies, giving intravenous glucose as the sole treatment raised the mortality rate above that for untreated control animals.\(^8\)\(^17\) The reasons for this are not totally understood.

More recent treatments have included the use of corticosteroids and/or ACTH to increase gluconeogenesis and thus increase the blood glucose levels.\(^16\)\(^18\) This treatment may be successful in early cases, although it may also induce parturition as a side-effect. In severe cases, however, this treatment is often unsuccessful. In fact, it may be of no benefit at all since many advanced cases show elevated endogenous steroid levels prior to treatment as a natural physiological response that occurs in an attempt to reverse hypoglycemia.\(^4\)

Another treatment, used with some success in early cases of pregnancy toxemia, is oral dosing with glycerol or propylene glycol, both of which are glucogenic substances. A suggested dosage is 200 ml of 50% glycerol solution twice daily until the appetite returns to normal.\(^5\) This can be fairly effective if the disease has been detected in the early stages before it has progressed to total anorexia and recumbancy. Because of this relative efficacy in early cases and because of the relative ease of administration by the layman, this is considered by many to be the treatment of choice.\(^3\)\(^6\)

Insulin has also been suggested as a treatment, either alone or in conjunction with glucose therapy, to aid in the utilization of any available glucose.\(^5\)\(^7\)\(^8\)\(^17\) Some early studies seemed to show favorable results, especially with the insulin-glucose combination, but this treatment has since fallen into disfavor and is rarely used at present.\(^17\)\(^10\)

In recent years, work has been done in the use of anabolic steroids, in particular trenbolone acetate, as another treatment for pregnancy toxemia. These products cause marked appetite stimulation which may be sufficient to reverse the disease in those cases precipitated by a period of anorexia. There is also some evidence which indicates that trenbolone acetate may be useful in causing reversal of signs even in moderately severe cases that have progressed to the stage of recumbancy.\(^19\) There is more work to be done in this area, but these preliminary findings appear promising.

A final treatment that is used with some success is inducing parturition either chemically or by surgical intervention.\(^10\) This often produces good results in moderate cases, because once the fetal drain on maternal glucose is gone, the ewe easily returns to a normal state of glucose metabolism and will recover com-
pletely, provided irreversible nervous system lesions have not already occurred. 13 This treatment is restricted to use within 1 week of the expected lambing date, unless the production of a viable lamb is inconsequential. The pregnancy may need to be terminated at any time in the course of the disease in order to save the dam. Chemical intervention is simpler and easier than surgical intervention, and parturition can usually be accomplished by a single intramuscular dose of 10–15 mg of dexamethasone or 15 mg of ECP. A major reason that none of these treatments is successful in severe, advanced cases of pregnancy toxemia is that the changes in the brain, although precipitated by hypoglycemia, become unresponsive to glucose and thus irreversible in the later stages. 14 Consequently, treatments aimed at restoring normal blood glucose levels may be successful in doing this and still be unsuccessful in reversing the changes that have occurred within the nervous system.

PREVENTION

Since treatment of pregnancy toxemia is often unrewarding, prevention of the disease is very important. If the majority of ewes are thin in late gestation, the best and easiest way to prevent pregnancy toxemia is to insure that these ewes are on a rising plane of nutrition during late pregnancy. 6 In those cases where the ewe is overweight at breeding or in midgestation, she should be placed on a reducing diet to get her into moderate condition by late gestation. 6 In those cases where the ewe is overweight at breeding or in midgestation, the best and easiest way to prevent pregnancy toxemia is to insure that the changes in the brain, although precipitated by hypoglycemia, become unresponsive to glucose and thus irreversible in the later stages. 14 Consequently, treatments aimed at restoring normal blood glucose levels may be successful in doing this and still be unsuccessful in reversing the changes that have occurred within the nervous system.

CONCLUSION

Pregnancy toxemia is an important disease in major sheep-producing areas. It contributes to economic loss both through death of affected ewes and their offspring and through substandard performance in ewes that survive the disease. The condition is difficult to treat successfully in the early stages and becomes nearly impossible to treat in the late stages, which makes prevention a very important consideration. The producer must be informed how to properly manage his flock during gestation. With proper management, the incidence of the disease will approach zero and it will cease to be an economically important entity in sheep.

REFERENCES


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