Available online at www.sciencedirect.com
ScienceDirect
Small Ruminant Research 76 (2008) 77–82
Small Ruminant Research
www.elsevier.com/locate/smallrumres

Reflections on the causes and the diagnosis of peri-parturient losses of ewes☆

V.S. Mavrogianni [a,*], C. Brozos [b]

[a] *Veterinary Faculty, University of Thessaly, PO Box 199, 43100 Karditsa, Greece*
[b] *School of Veterinary Medicine, Aristotle University of Thessaloniki, Thessaloniki, Macedonia, Greece*

Available online 19 February 2008

Abstract

Peri-parturient loss of a ewe is of particular financial significance to a farmer. Therefore, veterinary advise before lambing is crucial, in order to avoid such losses altogether. This paper can be used as a brief guide for peri-parturient losses in ewes. The most common causes of losses can be classified as follows; pre-partum: metabolic disorders (mainly pregnancy toxaemia or hypocalcaemia), septicaemia–toxaemia consequent to abortion, clostridial infections; during lambing: obstetrical problems (ringwomb, uterine or vaginal rupture) leading to genital traumas and haemorrhages; immediately post-partum: septicaemia–toxaemia consequent to acute metritis or mastitis, metabolic disorders (mainly hypocalcaemia), clostridial infections. A brief account of some of these conditions is presented. Finally, a guide to diagnosis of the cause of a loss is included.

Keywords: Sheep; Death; Lambing; Pregnancy toxaemia; Hypocalcaemia; Obstetrical interference; Mastitis; Metritis

1. Introduction

Annual rate of ewe losses (deaths) averages up to 5% in sheep flocks, although in some situations the percentage may rise up to 10% (Hindson and Winter, 1990). The majority of these losses occur peri-parturiently, i.e. at the time of lambing (before, during and soon after it). In fact, the peri-parturient period poses significant threats for the life of a ewe, as a result of the endocrinological, immunological and clinical changes.

Many causes of peri-parturient losses have been identified at lambing time, although the most common are obstetrical complications and metabolic diseases at late pregnancy, which have remained untreated. As a general rule, the most common causes of losses, directly related with the peri-parturient period, can be classified as follows.

- Pre-partum: metabolic disorders (mainly pregnancy toxaemia or hypocalcaemia), septicaemia–toxaemia consequent to abortion, clostridial infections.
- During lambing: obstetrical problems (ringwomb, uterine or vaginal rupture) leading to genital traumas and haemorrhages.
- Immediately post-partum: septicaemia–toxaemia consequent to acute metritis or mastitis, metabolic disorders (mainly hypocalcaemia), clostridial infections.

The possibility that a loss may be caused by an unrelated cause (e.g., pneumonia) should always be kept in mind. In these cases, however, the peri-parturient state might have facilitated the death of the animal by exerting an immunosuppressant effect.

☆ This paper is part of the special issue entitled "Current issues in Sheep Health and Welfare" guest edited by George C. Fthenakis.

* Corresponding author.
E-mail address: vmavrog@vet.uth.gr (V.S. Mavrogianni).

0921-4488/$ – see front matter © 2007 Elsevier B.V. All rights reserved.
doi:10.1016/j.smallrumres.2007.12.019

This paper briefly reviews the major causes of peri-parturient losses of ewes, as well presenting diagnostic procedures in those cases.

2. Specific causes of peri-parturient deaths

2.1. Pregnancy toxaemia

Pregnancy toxaemia is a metabolic disease of ewes, occurring during late pregnancy and with a high mortality rate (Caldeira et al., 2007). It occurs independently of age or breed of animals, but ewes carrying twin or triplet fetuses are more prone to develop the disorder (Firat and Ozpinar, 2002; Sargison, 2007). Poor nutrition of ewes, especially during late pregnancy (Liamadis and Mills, 2007), also predisposes to the disease. Teeth or feet problems, as well as high parasitic infestations (Papadopoulos et al., 2007) may also lead to the disease, consequent to the poor condition of animals. Obese or lean ewes are more likely to develop the disorder. The former, predisposes to the disease due to excessive fat storage around rumen, therefore reducing and finally eliminating dry-matter intake. Consequently, fat mobilization is enhanced and liver function is aggravated. The latter, could lead to the disease due to the inadequate energy reservoirs during a period of increased demand.

Ewes show hyperketonaemia and hypoglycaemia. The disease is associated with disturbances of fat and carbohydrate metabolism, which affect the function of the liver and the kidneys (Andrews, 1997). Affected animals usually are in poor condition and show depression, selective anorexia (initially eating only hay and straw, subsequently eating straw only and finally not taking any feed), separation from the rest of the flock; soon thereafter, they develop neurological signs: tremors of the head and the neck, wandering, excessive salivation, unusual head position, absence of menace reflex, blindness. Finally, the affected ewes become recumbent and then, coma and death are inevitable (Sargison, 2007). If the fetus(es) die(s) *in utero*, there may be a transient improvement of the condition of the ewe, although subsequent embryo autolysis would result in further deterioration of the ewe's condition. Diagnosis of the disease is based on development at late pregnancy and on clinical signs; confirmation is achieved on measurement of ketone bodies concentration in urine. Measurement of blood glucose concentration can support the diagnosis.

Treatment is usually successful only if initiated at the early stages of the condition (Andrews, 1997; Sargison, 2007). *Per os* administration of propylene glycol or other glycogenic preparations can be useful; administration of glucocorticoids to cause abortion can also help. However, the same treatments may fail if the disease is at an advanced stage and the general condition of the animal is poor. In general, the condition should be viewed more as a flock problem. Antiparasitic treatment of apparently healthy animals, increase of feed energy and special care (e.g., housing and individual feeding) to ewes with poor condition or bad teeth will help prevent further cases in the flock (Panousis et al., 2001). Proper health management results to subsequent restoration of fertility in the flock (Mavrogianni and Fthenakis, 2005).

2.2. Hypocalcaemia

Calcium requirements of sheep depend on age, growth and reproductive stage (Bickhardt et al., 1998; Sykes, 2007). Calcium deficiency is particularly common during the peri-parturient period, when it can cause parturient paresis, more commonly referred to as hypocalcaemia ("milk fever", "downer ewe syndrome", "lambing sickness"). Selection for milk yield in dairy flocks has led to increasing incidence of the disease, especially during the post-partum period. Poor or imbalanced nutrition during late pregnancy can increase the incidence of the disease. Unlike cattle milk fever which always happens at parturition, hypocalcaemia in ewes can occur from several weeks before until the 1st weeks after lambing. In the early stages, ewes show incoordination and muscle tremors without the tetanic spasms associated with hypomagnesaemia. Usually ewes remain recumbent with their hind limbs extended and soon after pass to coma. If the animals do not receive any treatment, they usually die.

Diagnosis is based on measurement of the calcium concentration in the blood serum and the rapid response of ewe to intravenous calcium administration. Nevertheless, special attention should be paid to the differential diagnosis between pre-partum hypocalcemia and pregnancy toxemia, as intravenous calcium administration can be fetal in cases of impaired liver function. In ewes suffering with parturient paresis, administration of calcium as 200 g L^{-1} calcium borogluconate (20%, w/v), is advised (Sykes, 2007). Treated ewes respond rapidly, but the frequent relapses require close inspection and further treatments.

2.3. Acute post-parturient mastitis

Mastitis is the inflammation of the mammary gland. Clinical mastitis is manifested with obvious changes in the mammary gland and its secretion, occasionally accompanied by systemic signs. The disease may occur at any stage of lactation or during the dry-period, but

for the purposes of this account, only the immediately post-partum condition is described.

The secretion can be abnormal (serous, serosanguineous, haemorrhagic) and the mammary gland swollen, oedematous, hot, painful, whilst the skin may be discoloured. The ewe may also show systemic clinical signs (fever, depression, anorexia, lameness). Occasionally, the disease can lead to death of the affected ewe. Specifically in the immediately post-partum period, the most common agent of the disease is *Escherichia coli*; other agents are *Staphylococcus aureus*, coagulase-negative staphylococci and *Mannheimia haemolytica* (Fthenakis, 1994; Watkins and Jones, 2007). The disease is usually associated with increased bacterial numbers in lambing pens (Sevi et al., 1999). Recent findings (Mavrogianni et al., 2007) have also indicated that bacteria persisting into the mammary gland from the previous lactation may cause a recrudescence of the disease immediately post-partum. These authors have suggested that, as peri-parturient relaxation of immunity occurs, post-partum mastitis may not necessarily be caused by newly infecting organisms, but from these pre-existing microorganisms, which invade the mammary parenchyma and cause clinical mastitis. Teat lesions have also been found to predispose to the disease (Mavrogianni et al., 2006; Mavrogianni and Fthenakis, 2007); a genetic background of susceptibility to the disease may also be important (Barillet, 2007).

Clinical diagnosis is based on macroscopic mammary changes. In order to identify the causative agent, a microbiological examination of the milk sample should be carried out. Early instigation of intramammary antibiotic administration is important for successful treatment of the disease, whilst flunixin can be used in the supporting treatment (McKellar, 2006).

2.4. Clostridial infections ("post-parturient gangrene")

Immediately post-partum, the external genitals of ewes may be infected by *Clostridium chauvoei*. Infection can occur and progress rapidly after parturition, if the region (vulva, vagina, perineum) is wounded during a difficult lambing, or consequently to obstetrical interference (Lewis, 2007). Then, the infected animal develops high fever, whilst the skin or the mucosa of the infected region may be discoloured. Subcutaneous oedema, particularly at the perineum, may be evident. Occasionally, a sanguineous, odorous vulvar discharge may be evident. The infection may extend to the thigh muscles, which become dark and swollen. During necropsy, similar lesions could be found at the vaginal or the uterine wall. *C. chauvoei* produces α-, β-, γ- and δ-toxins; these cause severe necrotizing myositis (if muscles are infected), toxaemia and death. After death, the carcass often swells, decomposes rapidly and pale yellow gelatinous fluid can be seen in subcutaneous tissues (Lewis, 2007).

Diagnosis is based on clinical and pathological findings. Vaccination of pregnant ewes is important and contributes to prevention of disease. Good hygiene during lambing, especially when obstetrical assistance is provided, also helps to minimize cases (Lewis, 2007).

2.5. Metritis

The genital tract is particularly susceptible to infections after lambing, as bacteria can invade therein (Noakes, 1996). However, the host usually can counteract, limit infection and prevent the disease. Nevertheless, in some cases various factors can predispose to development of acute metritis. These include: dystocia followed by obstetrical assistance, prolapse of the uterus, retained placenta, post-parturient ketosis (Tzora et al., 2002). In such cases, bacteria can colonize the non-involuting uterus, producing toxins which are absorbed and cause signs of toxaemia.

Ewes with metritis develop genital (swollen vulva and vagina, vaginal discharge, retention of fetal membranes) or systemic (anorexia, dehydration, fever, toxaemia) symptoms. If the condition remains untreated, it can lead to death. The bacteria implicated more frequently are *Arcanobacterium pyogenes* and *E. coli* (Tzora et al., 2002). Treatment should include an effective antimicrobial agent, oxytocin and non-steroid anti-inflammatory agents. Ewes respond rapidly to the treatment and usually, there are no consequences to their future fertility.

2.6. Traumas and injuries to the genital tract

Traumas and injuries to the genital tract are usually the consequence of unskilled intervention or of excessive interference by the farmer during lambing. These can result to uterine, cervical, vaginal or vulvar tearing or traumas (Hindson and Winter, 2007). Death can occur either as a consequence of severe haemorrhage or alternatively, from consequent development of metritis.

3. Procedures for investigation of peri-parturient deaths

The first question to be asked is when and which cases of peri-parturient losses should be investigated. One may set a rule by investigating the first ten cases that occur in a

given year, followed by a 20% sample of the subsequent cases within the same lambing period. There should be no acceptable rate of peri-parturient losses in a flock, because such cases are of particular financial significance to a farmer; actually, the farmer loses the "capital", as well as the expected "return" for the investment.

When investigating death incidents in adult ewes in the flock, the veterinarian should proceed by taking into account the following (Hindson and Winter, 1990).

- The history of the incident(s) and number of ewes which died.
- Details of health management of flock (vaccinations, antiparasitic treatments, nutritional regime, etc.).
- Previous deaths of ewes or other problems, during the lambing season in the previous year.
- Symptoms of the ewe(s) before death and symptoms in other animals.
- The exact time of death in relation to parturition (see below for estimation procedure).
- If available, results of paraclinical tests and gross pathological findings.
- Clinical findings in live, sick animals; a good clinical examination of affected ewes, as well as of other, clinically healthy animals may prove valuable.
- Any treatment(s) that the animals had received and their estimated efficacy.

3.1. Estimation of the time of death in relation to parturition

The shepherd should usually be able to confirm whether the ewe had lambed or not. Furthermore, this can be accurately confirmed by carrying out a post-mortem examination of the genital tract (Hindson and Winter, 1990).

If the cervix is closed and the uterus is gravid, then obviously the ewe died before lambing. One should also take into account that cases of fetal death and resorption can occur; in such cases, the cervix will be closed, but no gross fetal remains would be evident within the uterus. Alternatively, lambing might have started, accompanied by an incomplete dilatation of the cervix ("ringwomb").

If the cervix is open with presence of a dead embryo or fetal remains into the uterus or the genital tract, then obviously death occurred at the time of lambing.

If the cervix is open with absence of embryo within the uterus, then the ewe had recently lambed or aborted. One should take into account that by the 3rd to 4th day after lambing, the cervix is closed and uterine involution progresses rapidly thereafter (Noakes, 1996).

3.2. Post-mortem examination

If the animal died at the peri-parturient period, the post-mortem examination will be useful to determine accurately the time of death in relation to parturition; it will also be used to detect if there was any other disease in progress. As mentioned above, the possibility that death was unrelated to the peri-parturient period should always be in mind. There are specific points that the veterinarian should consider during a post-mortem examination. These depend on the period of death in relation to parturition.

If it is considered that the animal died before lambing, one proceeds by carrying out the following.

- Estimation of the body condition of the ewe.
- Examination of liver and of peri-renal and peritoneal fat (fatty liver or depletion of fat reserves indicative of pregnancy toxaemia).
- Sampling of urine (directly from the bladder) for estimation of presence of ketone bodies.
- Sampling of aqueous humour for measurement of calcium concentration (only if death occurred up to 24 h ago).
- Examination of placenta and condition of the fetus (separation, inflammation of the placenta, autolysis of fetus); has fetal death occurred before or after ewe's death?
- Examination of other organs of the ewe, in order to evaluate whether death was unrelated to the pregnancy (e.g., lesions in respiratory system may indicate death subsequent to pneumonia).

If it is considered that the animal died at lambing, one proceeds by carrying out the following.

- Estimation of the opening of the cervix ("ringwomb"?) and examination of fetus for malpresentation.
- Examination of the fetus for autolysis-emphysema (indicative of neglected dystocia) or for mummification and of the placenta for inflammation or necrosis (indicative of abortion).
- Examination of the genitals for haemorrhages and bruises, which are indicative of manual interference; examination of the uterus for ruptures.
- Examination of other organs of the ewe, in order to evaluate whether death was unrelated to the pregnancy (e.g., lesions in respiratory system may indicate death subsequent to pneumonia).

If it is considered that the animal died soon after lambing, one proceeds by carrying out the following.

- Examination of the genitals for haemorrhages and bruises, which are indicative of manual interference.
- Examination of the uterus for ruptures; there may be small ruptures, which are very difficult to observe; in that case, one should also check for haemorrhages in the peritoneum.
- Examination of the uterus for retained embryo, for retained fetal membranes or for evidence of metritis; Fthenakis et al. (2000) have defined retention of fetal membranes as the failure to expel the placenta within 6 h after lambing of the last lamb.
- Examination of the tissues for presence of gas and discoloration, characteristic of clostridial infection.
- Sampling for measurement of calcium concentration (only if death occurred up to 24 h ago).
- Examination of the mammary gland for mastitis; the mammary glands should be soft, without abnormalities in the parenchyma or in the teats.
- Examination of other organs of the ewe, in order to evaluate whether death was unrelated to the pregnancy (e.g., lesions in respiratory system may indicate death subsequent to pneumonia).

3.3. Flock investigation

Subsequent to examining the dead ewes, a flock investigation should also be carried out. The following protocols should be employed.

- If investigating deaths during pregnancy: define the exact period of pregnancy, when the majority of deaths occurred (early-, mid-, late-pregnancy); ask shepherd about feeding of animals and availability of water, especially in pregnant ewes; confirm latest antiparasitic treatment; enquire about abortions in the flock; score body condition of dead and live animal(s) and examine teeth; observe animals and pay special attention to presence of vaginal discharge (characteristic of abortion) and evidence of recumbency (characteristic of metabolic diseases).
- If investigating deaths during the lambing period, additionally to the above: examine dead ewe(s) to confirm whether lambing had started (presence of fetal fluids, placenta or fetal parts); ask shepherd if he had interfered in some way; investigate whether new personnel have been employed in the farm as lambing assistants examine dead ewe(s) for presence of genital trauma (vaginal prolapse, rupture) and pay special attention to the cervix (ringwomb, rupture of cervix or uterus).
- If investigating deaths after lambing period: ask shepherd if ewes had lambed normally or if there had been any interference; examine ewe(s) for signs of interference or discharge (indicative of metritis); examine vulva for swelling (indicative of clostridial infection) or signs of necrosis (indicative of *F. necrophorum* infection); confirm latest anti-clostridial vaccination; examine mammary glands for evidence of severe, acute mastitis; observe animals and pay special attention to evidence of recumbency (characteristic of metabolic diseases).

4. Concluding remarks

As mentioned above, peri-parturient loss of a ewe is of particular financial significance to a farmer. Therefore, veterinary advise before lambing is crucial, in order to avoid such losses altogether. The present paper can be used as a brief guide to diagnose the causes of peri-parturient losses in ewes.

References

Andrews, A., 1997. Pregnancy toxaemia in the ewe. In Practice 19, 306–312.

Barillet, F., 2007. Genetic improvement for dairy production in sheep and goats. Small Rumin. Res. 70, 60–75.

Bickhardt, K., Henze, P., Ganter, M., 1998. Clinical findings and differential diagnosis in ketosis and hypocalcaemia of sheep. Deutsche Tierarztliche Wochenschrift 105, 413–419.

Caldeira, R.M., Belo, A.T., Santos, C.C., Vazques, M.I., Portugal, A.V., 2007. The effect of body condition score on blood metabolites and hormonal profiles in ewes. Small Rumin. Res. 68, 233–241.

Firat, A., Ozpinar, A., 2002. Metabolic profile of pre-pregnancy and early lactaton in multiple lambing Sakiz ewes. 1. Changes in plasma glucose, 3-hydroxybutyrate and cortisol levels. Ann. Nutr. Metabol. 46, 57–61.

Fthenakis, G.C., 1994. Prevalence and aetiology of subclinical mastitis in ewes of Southern Greece. Small Rumin. Res. 13, 293–300.

Fthenakis, G.C., Leontides, L.S., Amiridis, G.S., Saratsis, P., 2000. Incidence risk and clinical features of retention of foetal membranes in ewes in 28 flocks in southern Greece. Pract. Vet. Med. 43, 85–90.

Hindson, J.C., Winter, A.C., 1990. Outline of Clinical Diagnosis in Sheep. Wright, London.

Hindson, J.C., Winter, A.C., 2007. Genital abnormalities, obstetrical problems and birth injuries. In: Aitken, I.D. (Ed.), Diseases of Sheep, 4th edn. Blackwell, Oxford, pp. 75–80.

Lewis, C.J., 2007. Clostridial diseases. In: Aitken, I.A. (Ed.), Diseases of Sheep, 4th edn. Blackwell, Oxford, pp. 156–167.

Liamadis, D., Mills, Ch., 2007. Significance of quality of truly digestible protein on performance of ewes at late pregnancy and early lactation. Small Rumin. Res. 71, 67–74.

Mavrogianni, V.S., Cripps, P.J., Fthenakis, G.C., 2007. Bacterial flora and risk of infection of ovine teat duct and mammary gland throughout lactation. Pract. Vet. Med. 79, 163–173.

Mavrogianni, V.S., Cripps, P.J., Papaioannou, N., Taitzoglou, I., Fthenakis, G.C., 2006. Teat disorders predispose ewes to clinical mastitis after challenge with *Mainnheimia haemolytica*. Vet. Res. 37, 1–17.

Mavrogianni, V.S., Fthenakis, G.C., 2005. Reproductive consequences of pregnancy toxaemia in ewes. Reprod. Domest. Anim. 40, 354.

Mavrogianni, V.S., Fthenakis, G.C., 2007. Case reports: clinical, bacteriological, cytological and pathological features of teat disorders in ewes. J. Vet. Med. A 54, 219–223.

McKellar, Q.A., 2006. The health of the sheep industry and the medicines to maintain it. Small Rumin. Res. 62, 7–12.

Noakes, D., 1996. The puerperium and the care of the newborn. In: Arthur, G.H., Noakes, D.E., Pearson, H., Parkinson, T.J. (Eds.), Veterinary Reproduction and Obstetrics, 7th edn. Saunders, London, pp. 171–182.

Panousis, N., Brozos, C., Fthenakis, G.C., Karatzias, C., 2001. Pregnancy toxaemia of ewes. Bull. Hell. Vet. Med. Soc. 52, 89–96.

Papadopoulos, E., Arsenos, G., Coles, G.C., Himonas, C., 2007. Gastrointestinal nematode infection pattern of Greek dairy goats reared under extensive husbandry conditions and treated with anthelmintics at different times during the year. Small Rumin. Res. 69, 68–73.

Sargison, N.D., 2007. Pregnancy toxaemia. In: Aitken, I.D. (Ed.), Diseases of Sheep, 4th edn. Blackwell, Oxford, pp. 359–363.

Sevi, A., Massa, S., Annicchiarico, G., Dell'aquila, S., Muscio, A., 1999. Effect of stocking density on ewes milk yield, udder health and microenvironment. J. Dairy Res. 66, 489–499.

Sykes, A.R., 2007. Deficiency of mineral macro-elements. In: Aitken, I.D. (Ed.), Diseases of Sheep, 4th edn. Blackwell, Oxford, pp. 363–377.

Tzora, A., Leontides, L.S., Amiridis, G.S., Manos, G., Fthenakis, G.C., 2002. Bacteriological and epidemiological findings during examination of the uterine content of ewes with retention of fetal membranes. Theriogenology 57, 1809–1817.

Watkins, G.H., Jones, J.E.T., 2007. Mastitis and contagious agalactia. In: Aitken, I.D. (Ed.), Diseases of Sheep, 4th edn. Blackwell, Oxford, pp. 99–105.