Understanding patho-physiology of retained placenta and its management in cattle-a review

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Abstract
Retention of Placenta (ROP) in animals has adverse effects on fertility and production. However, understanding the pathophysiology and causes of ROP is important for managing this disease. The hormonal processes that lead to normal placental separation are multifactorial and begin before parturition. A variety of risk factors, including early or induced parturition, dystocia, hormonal imbalances, and immunosuppression, can interrupt these normal processes and result in retention of the placenta. Systemic administration of antibiotics can be beneficial for treating metritis after ROP, but antibiotic administration has not been shown to significantly improve future reproduction. Use of immunomodulators reduces the uterine inflammation and infection but is not used routinely. Collagenase injected into the umbilical arteries of retained placentas specifically targets the lack of placenta proteolysis and might enhance placental release. However, such therapy is costly and its benefits in terms of improving subsequent reproductive function have not been evaluated. The present review will highlight some of the effective means of management of ROP.

Keywords: Retention of placenta, cattle, pathophysiology, management

Introduction
Fetal membranes or what is known as “placenta” is an essential organ for prenatal transfer of nutrients and oxygen from the dam to the fetus. It normally drops within short time post partum (within 8 hrs of parturition), if it is retained up to 12 hrs then it is called as delayed removal and if retained for more than 24 hrs of parturition then it is called as ‘Retention of placenta’ (ROP). Such retention creates a number of problems by allowing microorganisms to grow inside the uterus causing its inflammation, fever, weight loss, decreased milk yield, longer calving intervals and may result in an open cow during the next year and if the infection is so bad the animal may actually die. Due to retention of placenta animal may suffer from tetanus as tetanus organism is commonly found in feaces and soil which requires at least 1-3 months as long term therapy. Retention of Placenta causes great economic losses, mainly due to decreased milk yield and calf crop.

Incidence
The incidence in cows varies from 4.0-16.1%. However, it can be much higher in problem herds and also increases during summer with increased parity, milk yield in the previous seasons and following birth of male fetus (El-Malky et al., 2010; Ahmed et al., 1999) Abortions, stillbirths and twin calvings resulted in increased incidence rates of 25.9, 16.4 and 43.8%, respectively. Also El-Malky et al. (2010) demonstrated an incidence of ROP reaching 4.6% in buffalo-cows over three years of study.

Mechanism of fetal membrane separation
Three events are involved in cotyledon-caruncle detachment (Youngquist and Threlfall, 2007):

**Morphologic events:** Detachment of placenta in the cow involves separation of the finger-like cotyledon villi from the caruncle crypts without significant tearing of either fetal or maternal epithelia. For the cotyledon villi to separate from the caruncle crypt, it is critical that the mouth of the cotyledon “pouch” be opened first by proteolytic enzymes. This is achieved by formation of the “mouth” of the cotyledon toward the apex (dehiscence) or by following a concentric band pattern in which the edge of the cotyledon “pouch” is digested first.

After placental detachment is accomplished, uterine involution is completed in an average of 39 days in normal cows and 50 days in cows with ROP. By day 6 post partum, caruncle septa are disorganized; by day 15, caruncles are completely sloughed as a result of necrosis. Consequently, retained membranes are detached by caruncle necrosis within 6 to 10 days and not later than 17 days post partum. The surface of the endometrium is covered by new epithelium by day 26 to 30 post partum.

**Biochemical events:** Postpartum uterine biochemistry is dominated by increased collagenase and other protease activities that correlate with different stages of parturition, resulting in a massive breakdown of collagen and other proteins during uterine involution. As a result, the weight of the cow’s uterus decreases from 9.0kg at parturition to 1.0kg at 30 days post partum.

**Physiologic events:** Physiologic release of placenta is accomplished in most cows between 3 and 6 hours post partum. Cotyledon proteolysis (dehiscence) and decreasing adhesiveness (viscosity) of the cotyledon-caruncle interface fluids seem to be key factors in the release of placenta. Collagenases are capable of reducing the specific viscosity of collagen. Collagenase activity of cotyledon villi during delivery is increased in healthy cows and decreased in cows with ROP. The cellular sources of collagenase and proteolytic enzymes for placental release in the cow are unknown. In laboratory animals and humans, myometrial cells, fibroblasts, and leukocytes have been identified as sources of collagenase in the uterus. Lack of uterine motility is not considered as a reason for primary retention, because uterine motility is normal or above normal in cows with primary ROP. The direct cause of placental retention is uncertain, but it is related to a deficiency of myometrial contractions and failure of the maternal immune system to successfully degrade the placentomes at the end of pregnancy (Frazer et al., 2005).

**Failure of cotyledon-caruncle detaching mechanisms**

The key element in the pathogenesis of retained placenta is a failure of timely breakdown of the cotyledon-caruncle attachment after delivering the fetus. The most important risk factors for placental retention are abortion, stillbirth, twining, dystocia, induction of parturition with PGF$_2$α and caesarean section, metabolic disorders, especially milk fever. Prenatal losses can be caused by infectious and non infectious factors. Primary attention has been often directed to infectious causes, but non infectious factors probably account for 70% or more of the cases (Frazer et al., 2005). The infectious causes of placental retention are behind the scope. Non infectious causes are often multifactorial and are difficult to diagnose (Hanzen et al., 1999). The list of potential causes is quite long, however, there are a number of common causes (Ball et al., 2004).

**Hereditary causes of retained placenta:** Nasr et al. (1996) reported that cows having blood group genotype BGKOxA’0’ bred with sire have ‘I’ genotype dropped their placenta normally, while cows having BO$_2$Y$_2$ AE$_3$GP’ genotype bred with sire having genotype I$_2$ showing high incidence of ROP. On the other hand, the most frequent alleles in serum proteins of NROP (Non retention of placenta) cows were albumin (ALA), post albumin (PalA) and amylase (AmB) gene markers, while the most frequent genetic alleles in serum proteins of ROP cows were alpha globulin (F$_2$A) and transferrin D (TFD). Moreover, it was recommended to use the above mentioned genetic constituents of both dam and sire for breeding purposes In high lactating cows, somatotropins stimulate the production of insulin-like growth factor 1 (IGF-1) in the hepatic cells. However, the plasma IGF-1 level was found to be quite low, especially in the hyperketonaemic animals Huszenicz et al. (2006) in which metabolic products (non esterified fatty acids (NEFA) and βOH-butyrate (BHB)) accumulate and impairs the migration, phagocytic and killing activity and /or the oxidative burst of PMNs and other leukocytes, enhancing the susceptibility of host to invading pathogens (Reist et al., 2003).

**Mechanical causes of retained placenta:** Difficult birth (calf too large for cow, backward presentation of calf known as breech birth, one leg or head backwards), twins, late or premature birth, prenatal loss, induction of parturition with PGF$_2$α, cesarean
section and fetal monsters or emphysematous fetus (gas-filled fetus) are direct causes of dystocia and consequently to ROP.

**Nutritional causes of retained placenta:** Cows 0-120 days postpartum are at risk of ration formulation error and feed which leads to delivery problems. Periparturient dairy cows (0-20 days postpartum) undergo a transition from a relatively high fiber diet to a lactation diet that generally is higher in energy and lower in fiber. During this period, the amount of energy required for maintenance of body tissues and milk production usually exceeds the amount of energy in the diet (Goff et al., 1997). This nutrient deficit makes the cow susceptible to metabolic diseases such as ketosis and milk fever, which usually occur within 3 weeks of thereby calving slowing adaptation to the postpartum diet. Also, prepartum heavy grain feeding may be associated with both higher milk production and a higher incidence of left- displaced abomasum (Lucey et al., 1986) and increased risk of reproductive disorders such as dystocia, retained placenta, cystic ovaries, metritis Oetzel et al. (1995) prolapsed uterus Markusfeld et al. (1993) and acute clinical mastitis increasing the risk of non- parturient paresis (Gro et al., 1989) Cows being adapted to high- energy and high concentrate lactation rations are at risk because the rumen papillae need time to elongate and the microbial population needs time for adaptation. Also, lower dry matter intake near calving can result in depressed forage intake. It has generally been found that other feeding-related diseases increase the risk of non-feeding-related diseases rather than vice versa.

Vitamin and mineral deficiency conditions such as selenium, vitamin E and vitamin A, β-carotene and disturbed C/P (1.5/1) ratio can impair general immunity Sheldon et al. (2004) and may alter the competence of cellular self-defence mechanism and can increase the risk for placental retention and metritis. Ahmed et al. (2009) reported that ROP was associated with oxidative stress as shown by the obvious increase of blood malondialdehyde and nitric oxide and decreases of catalase, superoxide dismutase, ascorbic acid, glutathione reduced and total antioxidant capacity values with low zinc, copper, iron and selenium concentrations.

High milking cows with a greater degree of negative energy balance prepartum and higher NEFA concentrations were 80% more likely to suffer from ROP (LeBlanc et al., 2002). On the other side, over-conditioned cows were shown to be more sensitive to retained placenta and subsequent infertility than cows with normal body condition scores (Badinand et al., 1984). Etiological mechanism for retained placenta was associated with dietary-conditioned liver disorders and high plasma urea and gamma-glutamyl transferase concentrations (Lotthammer et al., 1983). Also low plasma glucose and PGFM (the mainPGF 2α metabolite) levels. Low monocyte and high red cell counts was traced in blood of cows with retained placenta, especially in the late gestation period Chassagne et al. (1992) and could be connected with the disease by means of dietary unbalanced ratio of n-3/n-6 polyunsaturated fatty acid (PUFAs) that would involve reductions in synthesis of cyclooxygenase products, impair uterine contractions, vascular tone and platelet aggregation leading to retained placenta (Michal et al., 2006).

**Hormonal imbalances:** Hormonal imbalance existing before delivery is effective in inducing retention of placenta. Placental separation occurs when foetal cortisol induces the production of the enzymes, 17-hydroxylase and aromatase in the placenta which favour oestrogen synthesis at the expense of progesterone synthesis. Maternal plasma levels of oestradiol-17 increase suddenly, while plasma levels of progesterone decline sharply immediately prior to parturition. It is supposed during the week before parturition, the level of estradiol reaches its maximum level to help the uterus to get rid of any remnant of fetal membranes. Therefore, a decreased level of estrogen may be indicated as a factor enhancing ROP (Chassagne et al., 1992).

Spontaneous myometrial contractility is augmented by autocrine and paracrine release of PGF 2α and parturition ensues. Disturbed endocrine function, high progesterone and cortisol levels and low oestriadiol level was traced in the blood cows with ROP (Michal et al., 2006). Increased progesterone level in ROP may be due to failure of the placenta to produce specific steroidal enzymes that help in progesterone aromatization and its conversion too estrogen (Ball and Peters, 2004). Hormonal imbalances existing before delivery are effective in inducing ROP. Progesterone, more than estrogen, inhibits uterine collagenases and slows uterine involution. Dexamethasone increases synthesis and utilization of progesterone by cotyledon tissues in the cow. These changes may contribute to blocking postpartum expression of cotyledoncollagenases. Moreover, it has been found that glucocorticoids down-regulate collagenases (Youngquist and Threlfall, 2007)

**Failure of maternal immune response:** Maternal immunological recognition of fetal MHC class I proteins expressed by trophoblast cells triggers an immune/inflammatory response that contributes to placental separation (De-Mouzon et al., 2006). This lymphocytic activation was suppressed at the foeto-
migratory activity (Engler 1994). The increased cortisol concentrations in cows have been proposed as factors in the pathogenesis of ROP in cattle. In fact, circulating neutrophils from cows with ROP produced less superoxide anion than did neutrophils from control cows. Positive chemotaxis resulted in an ROP incidence of 2.6%, and negative chemotaxis, 35.6%. Moreover, leukocytes are a mobile source of collagenases and may be involved in uterine regression and release of placenta (Youngquist and Threlfall, 2007).

Managemental causes: Myometrium contractility is the third component of self defence mechanisms, since uterus contractions expel the uterine content. Lack of exercise and hypocalemia are the most frequent causes of decreased myometrium contractility Sheldon and Dobson, (2004). However, in a study by Bajcsy et al. (2005) there was no correlation between blood ionized calcium (Ca$^{2+}$) concentrations and any of the contractility parameters. Also, the lack of uterine motility plays little or no role in the occurrence of retained placenta. Moreover, cows with retained placenta have normal or increased uterine activity in the days after calving (Frazer et al., 2005). Stress (Transportation, rough handling, poor feed conditions, Isolation from group, Lameness,) results in elevated corticosteroids and increased risk of placental retention.

Retained fetal membranes: “Living Organ”

When membranes are kept in controlled laboratory conditions, however, they are capable of active utilization of oxygen and glucose, and more than 30% of the cells can exclude vital dye for 3 or more days. When membranes are kept at 1°C to 2°C, they stay metabolically active for 8 weeks or longer. Fetal membranes have an outstanding potential to “survive” without a live fetus. These properties suggest that ROP may respond to ischemia, anoxia, and bacteria by releasing biochemicals that cause inflammation, thus predisposing the cow to metritis (Youngquist and Threlfall, 2007).

Economic and reproductive impact of retained placenta

Metritis: ROP and metritis are positively correlated. Cows with ROP had a significantly higher incidence of metritis (53%) than cows without ROP (30%); also, a significant difference was found between conception rates in cows with ROP and metritis (66%) and in those with only metritis (77%) (Youngquist and Threlfall, 2007). It has been proposed that the metritis that accompanies ROP results from the presence of decomposing placental tissues, which provide a
favorable environment for bacterial colonization. Coliform bacteria and high concentrations of endotoxins present in lochia of cows with ROP are potent inducers of prostaglandins and cytokines, favoring development of uterine infections (Dohmen et al., 2005). Bacteria found in the early postpartum uterus or their endotoxins may interact with retained membranes to secrete PGE2, which may further predispose the uterus to infection.

**Mastitis:** Although the main economic impact of ROP seems to be decreased milk production (more days open, decreased milk volume, milk from treated cows withheld), the correlation between ROP and mastitis is controversial. Milk from cows with retained placenta is unfit for human consumption and therefore cannot be sold. The fertility of dairy cows is affected when most cows in the herd suffer from retained placenta. This causes a direct loss to the farmer due to delayed calving leading to a lengthy period between births (calving intervals) and hence low milk production. It is unhygienic to milk a cow with decomposing afterbirth hanging on it (LeBlanc et al., 2002). The financial losses due to retained placenta in dairy cattle were due to its non-removal, increased culling rate, loss of milk production and the costs of veterinary treatment and drugs.

**Treatment for ROP**

**Objectives:** The treatment objectives for ROP are to cause early detachment of the membranes in order to reduce the occurrence of metritis, decrease milk losses, reduce reproductive inefficiency, and decrease veterinary expenses.

**Managemental aspects:** As the non infectious causes of placental retention is multi-factorial and difficult to be diagnosed, so special care should be paid for control measures rather than treatment protocols. The genetic aspect should be put in consideration to select animals having the minimal probability for the occurrence of ROP (Ahmed and Zaabal, 2009). Selection of bulls with suitable birth weight for the breed is essential to protect the herd from calving problem (Skidmore and Loskutoff, 1999).

**Nutrition:** Supplementation with balanced vitamin and mineral mixture in prepartum period is considered a prophylactic step to avoid fetal membrane retention. Prepartum supplementation with antioxidants, vitamin E (DL -tocopherol acetate, 1100 IU) and Se (sodium selenite, 30 mg) by single I/M injection, at 3 week prepartum, is used as a prophylactic dose to avoid placental retention in cows (Gupta et al., 2005).

**Manual removal:** Manual removal of retained membranes is contraindicated because uterine infections are more frequent and more severe after this form of intervention. Bolinder et al. (1988) found that manual removal prolonged the interval from calving to 1st functioning CL by 20 days. The removal of an attached placenta causes damage to the endometrium and suppresses uterine leukocyte phagocytosis, Vandeplaschse et al. (1982) both of which encourage bacterial invasion (Peters and Laven, 1996). It is also not easy to properly remove a retained placenta; 62% can be removed completely, 27% partially, and 11% are non-removable. Often, attempts at removal during the first 48 hours after calving are unsuccessful because the placenta is too firmly attached and the apical part of the gravid horn is beyond the reach. It is also strictly prohibited to remove the retained placenta manually at high temperature.

**Hormones:** Hormones are not effective in either detaching the placenta in primary ROP or preventing early postpartum metritis in cows with ROP. Induced labor associated with the incidence of ROP was also reduced when relaxin was administered along with dexamethasone or cloprostenol Musah et al. (1986) presumably because of relaxin promoting collagenase activity that could counteract the inhibitory effects of dexamethasone. But some of the hormones are used for contraction of uterus thereby, expulsion of fetal membranes. The commonly used hormones are:

**Prostaglandin:** PGF-α does not cause detachment of retained membranes, but can improve reproductive performance in the early postpartum cow due to uterokinetic effect (Youngquist and Threlfall, 2007). Induction of labor with dexamethasone, with or without prostaglandin, is an established risk factor for ROP in cattle, although the exact mechanism for this is unclear (Gross et al., 1985). It has been suggested that glucocorticoids could have a direct inhibitory effect on collagenase activity (Guerin et al., 2004). Also, dexamethasone inhibits PGF$_2α$ synthesis within cotyledon cells, Gross et al. (1986) and administering prostaglandin along with dexamethasone reduces but does not eliminate the occurrence of ROP (Gross et al., 1985 and 1986).

**Oxytocin:** Oxytocin is the uterokinetic hormone of choice in the early postpartum cow. 20 IU three to four times daily have been used for ROP (Youngquist and Threlfall, 2007).

**Antibiotics:** The use of antimicrobial therapy in the treatment of ROP has demonstrated conflicting results (Peters and Laven, 1996). Postpartum metritis is a...
common sequela of ROP, and the rationale behind antibiotics for ROP is to prevent or treat metritis and its subsequent negative effects on fertility. One opinion is that antibiotics should not be used to treat ROP because they may delay the release of retained membranes by inhibiting the putrefaction process. Local antimicrobials, typically given as uterine infusions or boluses, have not been shown to reduce the incidence of metritis or improve fertility (Peters and Laven, 1996).

In one study (Youngquist and Threlfall, 2007), despite intrauterine treatment with 5g of tetracycline powder on day 1 and 10.5 million U of procaine penicillin G IM on days 1, 2, and 3, 76% of the cows developed metritis. The reason for the lack of effectiveness of antibiotics has been attributed to the use of inadequate dosages, because it is difficult to attain adequate tissue concentrations of antibiotics in the uterus by using uterine infusions. Intrauterine tetracycline has been reported to have a negative effect on subsequent fertility. A possible explanation for this effect is that tetracycline may cause irritation of the endometrium and perhaps also of the metabolically active retained membranes. Chemical inflammation may override the beneficial effects of the antibiotic. Another complicating factor with infusion of antibiotics into the uterus is the extreme pH of the antibiotic solutions used, which may contribute to damage of the uterus. For example, tetracycline solutions can be strongly acidic (with a pH of 1.8–3.0) or alkaline (for Oxytetracycline for IV injections, pH is 8.0–8.5). Nevertheless, there is consistent support for the use of antibiotics, especially in severe cases of metritis, provided that adequate dosage and route of administration are used.

Systemic antibiotics are believed to be beneficial in ROP cases where fever was also present (Drillich et al., 2006 and LeBlanc et al., 2008). Systemic antibiotics alone were just as effective as systemic antibiotics combined with intrauterine treatment (Drillich et al., 2006). However, because all febrile cows were treated systemically, it is unclear whether the resolution of fever was caused by the antibiotics or to the cow’s own immune defense mechanisms. There are currently no known trials in which cows with ROP and fever were left untreated (LeBlanc et al., 2008). Treating all ROP cows with systemic cefiofur regardless of temperature was not superior, in terms of occurrence of fever, shedding of retained fetal membranes, or subsequent reproductive parameters, to selective antibiotic treatment of only febrile cows (Drillich et al., 2006). Treating RFM for 5 days with 2.2mg/kg of systemically administered cefiofur was superior in preventing metritis when compared with estradiol or no treatment, although no significant improvements in reproductive performance were found (Nadja et al., 2007). LeBlanc, (2008) stressed that at present it seems economically preferable to selectively treat metritis cases rather than automatically treat all cows with retained placenta with antibiotics.

**Immunomodulators:** E Coli LPS plus oxytocin effectively reduced the uterine inflammation and infection, thus increasing the overall reproductive efficiency in chronically sub fertile mares. LPS is a potent secretagogue for a variety of inflammatory mediators and immunoregulatory cytokines from endometrial cells and leukocytes. The failure to produce any systemic endotoxic responses after intrauterine infusion of E coli endotoxin confirms that E coli LPS can be used safely without producing any adverse reaction in mares (Nadja et al., 2007). Recombinant human interleukin 8 (rhIL-8) was effective in attracting PMNs into the uterus within 6 hours after administration in cattle and horses (Zerbe et al., 2003).

**Antiseptics:** A variety of antiseptics, including chlorhexidine and dilute iodine, have been used in the treatment and prevention of ROP. In most cases, their efficacy remains to be demonstrated. These compounds should be used with caution, especially iodine preparations, some of which can be extremely irritating.

**Collagenase:** A new approach for the treatment of ROP is the injection of collagenase into the umbilical arteries retrieved from ROP. Bacterial collagenase from Clostridium histolyticum is used because it can degrade several types of collagen, it is commercially available, it is affordable, and it does not cause residual blood clotting in placenta. Collagenase therapy shows promise as a means of treating retained placenta in a variety of species (Eiler et al., 1993, and Haffner et al., 1998).
perfusion of the entire placenta, a volume of 1L is injected. Oxytetracycline (100mg total dose, which is approximately 30mg/kg fetal membranes) for intravenous injection can be added to 1L of collagenase solution if an antibiotic is desired; however, final pH of the solution should be adjusted to approximately 7.5. Five hundred ml of collagenase solution is injected into each artery or 1000ml into one artery if only one is available. Because of arterial anastomosis, it is unnecessary to inject the two arteries in single births or the two umbilical cords in twin births. Thirty-six hours later, the retained membranes are easily extracted by gentle traction if they have not been expelled spontaneously (Eiler et al., 1993). This treatment is safe and has no side effects. It can be applied between 12 hours (best) and 96 hours after calving. After 48 hours of retention, there is a tendency for the residual blood in the placenta to clot and for anastomoses to close, which limits the perfusion of retained membranes with collagenase solution. The technique is simple, and the procedure can be completed in 25 minutes by a skilled veterinarian without an assistant. Oxytetracycline plus collagenase constitutes a useful, therapeutic combination, owing to the advantage of loosening retained membranes while preventing infection. Antibiotic collagenase combinations should be used with caution, however, to avoid inactivation of collagenase by the antibiotic.

Herbs and herbal preparations:
- Garlic: It has cleansing action and helps in expulsion of retained fetal membranes.
- Feeding of bamboo leaves mixed with oil bran or bajra.
- Feeding of parched chick pea floor (1 kg) mixed with butter milk (rabri).
- Feeding of electuary prepared from liquid extract of ergot (8 ml), Quinine sulphate (4g)
- Magnesium sulphate (200g), pulv.Gentian (16g) and molasses.
- Herbal preparations like Replanta Powder, Utrefit, and Uretone can also be used for treatment of ROP.

Prevention of retained placenta

References

Current recommendations for prevention of RFM in cattle include cow comfort, reducing stress around parturition, and careful nutritional management, particularly during the transition period. Due to metabolic diseases uterine immunity is impaired Zerbe et al. (2001) so proper nutrition in prepartum period is provided to avoid metabolic diseases. Vitamin and mineral deficiencies can impair general immunity, to avoid this Vitamin E and Selenium supplementation should be given Bourne et al. (2007) and of Ca:P ratio of 1.5:1.0 should be maintained and P should be supplemented. The infectious diseases can be prevented by proper immunization against specific infection

Conclusion
Placental detachment and expulsion are complex processes that begin with prepartum hormonal and biochemical changes. Disturbances in any of these normal processes may result in placental retention. An understanding of the physiology of placental retention allows discussions about the link of risk factors to specific causes that will aid in the critical appraisal of treatment protocols and prevention of bovine ROP. Thus, ROP can be thought of more as a syndrome with possibly multifactorial causes as well as reflecting herd management. Many common therapies for ROP have not been shown to be effective, and some could actually have a negative impact on future reproduction. Manual removal, local antibiotics, and prostaglandins are used treatments, although current evidence does not support their use. When cows become febrile, systemic cefiofur has been the most widely evaluated antibiotic, and appears to be beneficial in reducing disease and aiding in the return to normal reproductive function. Collagenase might prove to be valuable in achieving faster release, although cost prohibitive in many cases. New therapies should be aimed at correcting specific causes of ROP. The limited availability of effective treatment options emphasizes the importance of prevention. Current recommendations for prevention of ROP in cattle include cow comfort, reducing stress around parturition, and careful nutritional management, particularly during the transition period. Supplementing vitamin E and selenium may be an effective preventive measure.

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