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Review Paper

A Clinical Approach to the Diagnosis and Treatment of Retained Fetal Membranes with an Emphasis Placed on the Critically Ill Mare

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ABSTRACT

Retained fetal membranes (RFMs) in mares can be defined as the failure of partial or complete release of the allantochorion by 3 hours after parturition. The incidence of RFM ranges from 2% to 10% of foalings in light breed-type mares, and it has been reported to be as high as 30%-54% in Friesian mares. This peripartum problem occurs specially after dystocia, prolonged gestation, cesarean section, fetotomy, hydropsy, and induced delivery. Uncomplicated abortion, stillbirth, and twinning are not necessarily associated with RFM, unless dystocia also occurs. Occasionally, RFM can occur after an apparent normal foaling for unknown reasons. Many predisposing factors have been postulated: uterine inertia and fatigue, calcium/phosphorus imbalance, selenium deficiency, abnormal hormonal environment, physical mechanical intervention, fescue toxicosis, aging, individual predisposition, and placentitis. This condition can lead to severe problems in mares, including metritis, laminitis, and death. The treatment for this condition can be addressed at three different major levels: uterine clearance, control of shock and endotoxemia, and treatment and prevention of laminitis. This manuscript reviews some of the most relevant articles addressing this reproductive problem and offers the authors' clinical experience on dealing with RFM.

1. Introduction

Retained fetal membranes (RFMs) is the most common postpartum complication in mares [1,2] and is characterized by a partial or complete failure of the allantochorion to detach from the endometrium [3]. Typically, expulsion of fetal membranes occurs shortly after foaling, and it is considered retained if it is not expelled within 3 hours postpartum [1,2,4-7]. The prevalence of RFM ranges from 2% to 10% of foalings [8], and it has been reported to be as high as 30%-54% of uneventful parturitions in Friesian mares [9].

RFMs often occur after dystocia, prolonged gestation, cesarean section, fetotomy, hydropsy, induced delivery, and periparturient hemorrhage [6,8,10-12]. Abortion, stillbirth, and twinning are not associated with an increased risk of RFM, unless these conditions occur concomitantly with dystocia [10]. Occasionally, RFM occurs after an apparently normal foaling for unknown or unidentified reasons [2].

Many factors have been postulated to predispose the mare to the development of RFM, including uterine inertia and fatigue, selenium deficiency, calcium/phosphorus

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imbalances, abnormal hormonal environment, physical (mechanical) intervention during foaling (eg, dystocia), fescue toxicosis, placentitis, and advanced age [2,4-6]. Age does not appear to be a predisposing factor in Draft mares [4]. It has been reported that mares with a history of RFM have a higher probability of recurrence when compared with mares with no history of RFM [10].

This puerperal abnormality (ie, RFM) is a significant health risk to the mare and a financial burden to the horse breeder. Thus, the condition should be diagnosed quickly and appropriate treatment should be administered promptly to enhance the likelihood of a positive outcome and to prevent secondary life-threatening complications. Based on a review of the literature in combination with relevant clinical experiences, here, we describe the diagnosis and treatment of RFM, with special emphasis placed on the critically ill mare.

2. Pathophysiology of RFMs

The pathophysiology of RFM in mares has not been completely elucidated. After rupture of the umbilical cord at parturition, the vessels in the umbilicus and fetal membranes collapse and the blood flow to the allantochorion is reduced, causing a reduction in the blood volume of the microcotyledons, resulting in separation of the chorionic villi from the endometrial crypts [2,13]. These events, along with postpartum uterine contractions moving from the tip of the horns toward the cervix, facilitate detachment and eventual invagination of the membranes into the horn, so that the fetal membranes are normally expelled inside out. Expulsion of the fetal membranes is also facilitated by the gentle traction that their physical weight imposes as it protrudes out from the vulvar lips.

Retention of fetal membranes occurs most commonly at the tip of the nonpregnant horn [6,8] (Fig. 1). It has been hypothesized that this is owing to the greater interdigitation of the microvilli (microcotyledons) at the tip of the nongravid horn when compared with the edematous pregnant horn, where the microvilli are more compressed and smaller [8]. The increased prevalence of RFM after dystocias may reflect greater edema and microhematoma formation in the microcotyledons that resulted from physical manipulation of the membranes [2]. In cases of RFM associated with induced and/or premature foaling (eg, cases of complicated abortions, periparturient hemorrhage, colic, prepubic tendon rupture, etc), the lack of the necessary hormonal signals or patterns may not allow postpartum fetal membranes' release [2,6,12]. Absent or weak uterine contractions associated with uterine inertia (primary or secondary) are also a potential cause of RFM because of the decreased expulsive myometrial efforts [2]. Electrolyte imbalances have also been suggested to cause RFM.

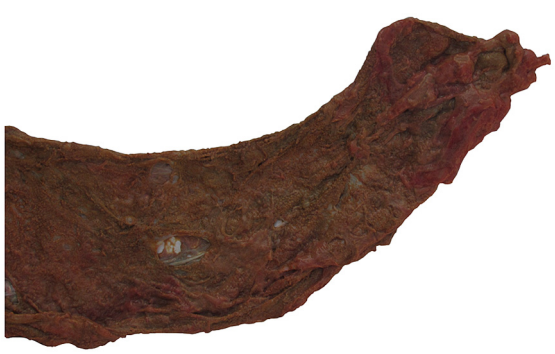

Fig. 1. Nongravid horn fetal membrane (ie, chorioallantois) retention after uneventful parturition. This necrotic segment was retained for 4 days.

A retrospective study of 155 Friesian broodmares with normal deliveries found significantly lower serum calcium concentrations 12 hours postpartum (12.30 mg/dL) in mares with RFM compared with mares that expelled the fetal membranes within 3 hours after normal foaling (12.72 mg/dL) [9]. These investigators also reported that a greater number of mares with RFM responded to treatment with a combination of oxytocin and calcium−magnesium borogluconate solution as compared with mares suffering from RFM and treated solely with oxytocin in saline solution. This study highlights the benefits of calcium supplementation even if serum calcium values are within normal ranges. However, it is worth noting that the study by Sevinga et al. [9] did not evaluate ionized calcium, which is physiologically active.

Preexisting fetal membranes' pathology may also increase the risk of RFM. Histological examination of the fetal membranes from 88% of Draft mares suffering from RFM demonstrated fibrosis and excessive connective tissue development in the allantochorion (villi and stromal tissue), with enlarged allantochorion epithelial cells and sparse and a lower number of branched allantochorion villi [14]. However, at this time, it is difficult to state whether these histological findings are the causes or results of RFM; therefore, their role in the pathogenesis of RFM needs to be clarified.

3. Diagnosis

Diagnosis of RFM may be obvious in mares in which the fetal membranes are protruding from the vulva (Figs. 2-4), whereas it is more challenging when a small piece of fetal membrane is retained within the uterus (Fig. 5). Thus, postpartum fetal membranes' examination is paramount to assess intactness and normal morphology (Fig. 6) and for early diagnosis of retention of portions of the fetal membranes within the uterus (also called "placental tags") (Fig. 5). In cases in which the fetal membranes are not available for inspection, ultrasonographic evaluation of the uterus per rectum is used to detect hyperechoic structures in the lumen or within the uterine endometrium [15].

Early diagnosis of RFM is critical to prevent severe and potentially fatal complications such as metritis, septicemia, and laminitis [13], which can start to develop as early as 4-8 hours after delivery of the foal [2,6]. Necrotic fetal membranes within the uterus provide an excellent environment for bacterial growth and endotoxin production and release. Systemic absorption of endotoxin and bacteremia are facilitated because of the inflammation and already-present normal pre- and postpartum uterine vessels' dilation.

In cases where RFM is not diagnosed early, mares may present with fever, colic signs, and odorous vaginal discharge, which is a result of development/progression to metritis. If not treated, they will progress to septicemia/endotoxemia,