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Monitoring of the last third of gestation and peripartum disorders

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Abstract

Pregnancy is a dynamic process that extends from conception to birth. Optimal conditions during this period of life are necessary to ensure successful intrauterine fetal development and to guarantee survival capacity of the offspring in the external environment. Thus, the improvement of current models of gestational and neonatal assessment, and development of new methodologies should aim to predict the outcome of pregnancy for both mother and newborn. This review aims to explore with a logical and sequential approach, the main diseases that affect the pre , intra and postnatal periods in the mare, so that the prevention or reduction of perinatal injuries can be achieved successfully.

Keywords: birth, disordes, mare, peripartum, pregnancy.

Introduction

How to assess the mare during the peripartum period?

Traditionally, the majority of pregnant mares are gynecologically unsupervised from part of the second trimester until the end of pregnancy, however, the feto-placental unit should be monitored throughout pregnancy to collect biochemical and biophysical parameters in order to reduce and prevent perinatal insults (Bucca, 2006).

The mare´s medical and reproductive history should be evaluated, including data regarding injuries at birth and medical problems. The complete physical examination of the pregnant mare should also be performed, including rectal palpation and biochemical and hematological parameters that demonstrate the female reproductive tract's health status. The rectal examination includes palpation of the cervix, uterus, fetus and all abdominal structures, as the vaginal examination should be done with discretion to avoid the development of iatrogenic ascending placentitis contamination (Wilkins, 2006).

Biochemical parameters are still under investigation and are primarily used in the evaluation of fetal compromise and to predict the neonatal adaptability. Thus, evaluations such as estrogen (Riddle, 2003) and progestogen (Rossdale *et al*., 1991; Rossdale, 2004; LeBlanc *et al*., 2004) hormone profiles, determination of the concentration of electrolytes in the mammary gland (Paccamonti, 2001), colostrum density (Jeffcott, 1974; LeBlanc, 1984), and occasionally fetal fluid analysis (Schimidt *et al*., 1991; McGladdery *et al*., 1992) can be performed. The fetal fluid analysis enables the determination of prostaglandin concentration, assesses karyotype and fetal cytology to detect evidence of infection, however, amniocentesis and alantocentesis are often not performed due to the potential risk of abortion. A good alternative is the collection of fetal fluids for viral and bacterial culture during delivery with no risk to the mare and to the fetus (Bucca, 2006).

The biophysical parameters can be evaluated with the B-mode and Doppler-mode ultrasound, both transrectally and transabdominally, and still by electrocardiogram. Fetal static can be evaluated (Ginther and Griffin, 1993; Bucca *et al*., 2005), fetal heartbeat and rhythm (Adams-Brendemuhel and Pipers, 1987), activity (Fraser, 1975) and fetal size, stomach measurement (Bucca *et al*., 2005), and fetal bladder (McGladdery *et al*., 1993). Uteroplacental thickness and contrast evaluation, quantity and echogenicity of fetal fluids (Renaudin *et al*., 1997; Bucca *et al*., 2005), and multiple gestations can also be evaluated by ultrasound. Ultrasonography and electrocardiogram can be performed continuously to detect changes in the fetus, uterine stress and neonatal hypoxia (bradycardia) and tachycardia due to fetal movements or when tachycardia related to fetal stress is persistent (McGladdery *et al*., 1993).

Placentitis

The changes of the placenta are not easily recognized and any dysfunction represents a difficult diagnosis. Placentitis could affect utero-placental contact and placental efficiency, and consequently fetal well-being (Bucca, 2006).

The placentitis is an important factor of abortion and perinatal death (9.5-33.5%) in the equine species (Giles *et al*., 1993; Smith *et al*., 2003). Different etiological agents such as viruses (equine viral arteritis and herpes virus type 1), bacteria (β-hemolytic *Streptococcus*, *Eschericcia coli*, *Pseudomonas aeruginosa*, *Klebsiella pneumoniae*, *Leptospira spp* and *Crossiella equi*; Donahue *et al*., 2002) and fungi (*Aspergillus spp* and *Mucor spp*) may be involved, and different infection routes are possible (Bucca, 2006). Fifty-three percent of the losses are due to bacterial infections and in 28% of these cases *Streptococcus zooepidemicus* was isolated (Giles *et al*., 1993). The ascending infection route is the most common and results in inflammation and early detachment in the cervical star region of the placenta and may be present

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in mares with pertineal conformation defect, abnormal cervical and vaginal examination history during pregnancy. The hematogenous infection route is commonly used by the bacteria *Leptospira spp*, which causes a diffuse or multifocal placentitis. The *Crossiella equi* in turn infects the uterus at the time of breeding, but only promotes placentitis in late pregnancy and presents characteristic lesions in the uterine body (Bucca, 2006).

Placentitis clinical signs are presented as early mammary gland development with production and loss of colostrum/milk, cervical relaxation, vaginal discharge, abortion, premature labor, premature/dysmature foal, small neonates with sepsis and stillbirth. Recent studies suggest that chorioallantoic infection results in increased expression of inflammatory mediators associated with local effects that alter myometrial contractility and compromise fetal viability (McGlothlin *et al*., 2004). According to Lyle and Paccamonti (2010), stress in uterus promoted by infection appears to accelerate hypothalamic-pituitary-adrenal axis maturation of the fetus, leading to miscarriages and premature births.

Systemic changes in blood profile are not always present and clear. Clinical signs associated with cytologic evaluation, cervico-vaginal discharge culture, along with the placental ultrasound evaluation determine the placentitis diagnosis (Bucca, 2006).

In ultrasound evaluation the combined thickness of uterus and placenta can be measured, and the reference values are <5 mm (<270 days), <8 mm (271-300 days), <10 mm (310-330 days) and <12 mm (>330 days) (Renaudin *et al*., 1997; Bucca, 2006). Inflammation signs such as an increased thickness of uterus and placenta, which can be measured mainly in the cervical star and uterine body regions, edema of the chorioallantois and early placental detachment, might be detected.

Before the bacterial culture and sensitivity test results are ready, the treatment must be started with a broad-spectrum antibiotic that crosses the uterus-placental barrier and have good distribution in fetal fluids. Nonsteroidal anti-inflammatory drugs also should be used to decrease mediators present in inflammatory placentitis and progestogen supplementation may assist in the maintenance of uterine quiescence (Bucca, 2006). The progesterone administration concept is still controversial. At 100 days of gestation this procedure could be interesting to maintain pregnancy in mares suffering luteolysis mediated by PGF2α production (Daels *et al*., 1991). In more advanced pregnancies, if the placental unit is committed to progesterone production, this possibly will be compromised in relation to fetal nutrition and oxygenation, and may lead to the dead fetus retention as described by McCue *et al*. (1997). Treatment with progestins has long been advocated to promote uterine quiescence in mares with uterine pathology. The actual rationale for progestin use in late pregnancy is not clear. Presumably, the anti-prostaglandin effect of progestins would contribute to reduced myometrial activity by interfering with up regulation of prostaglandin and oxytocin receptors (Garfield *et al*., 1980). Without receptor expression, gap junction formation would be inhibited and uterine contractility prevented. Daels *et al*. (1996) tested the effect of progesterone and altrenogest (a synthetic progestin) on pregnancy maintenance, in mares treated with the prostaglandin analog cloprostenol, and demonstrated that progestin supplementation was able to prevent prostaglandin-induced abortion in most cases supporting progestin supplementation use in mares at risk for preterm labor. Similarly, Frazer *et al*. (2002) and Bucca (2006) indicated that progestogen supplementation might be used on cases in which it is possible to evaluate the pregnancy in regular intervals.

Fetal membranes hydrops

The hydramnios and hydrallantois are exaggerated fluid accumulation in the amniotic and allantoic compartments respectively, being hydrallantois the most common disorder. These conditions are rare in the horse, and the prognosis for pregnancy is considered reserved (Vandeplassche *et al*., 1976; Koterba *et al*., 1983; Waelchli and Ehrensperger, 1988). Hydrallantois is related to mild diffuse placentitis or endometrial vasculitis, while hydramnios has unknown etiology (Koterba *et al*., 1983) and may be due to defects in the fetus swallowing system (Bucca, 2006).

Fetal membranes hydrops can occur in multiparous and 6-20 yr old mares, generally in the last trimester of pregnancy (Vandeplassche, 1987). Typically there is a sudden onset (1 to 2 weeks), abdominal distension associated with varying degrees of colic and defecation difficulty (Bucca, 2006). There is progressive appetite loss, difficulty to walk and dyspnea associated with cyanotic mucosa especially in recumbent mares (Vandeplassche, 1987).

On physical examination, rectal temperature is normal; however, the pulse is high. On rectal palpation, with extreme care and lubrication, one can perceive high pressure due to the large accumulation of intrauterine fluid. Feces tend to be covered with mucus due to slow passage through the gastrointestinal tract (Frazer *et al*., 1997b).

The normal allantoic fluid volume in mares at term varies from 8 to 18 liters. However in documented hydrops cases it varies from 110 to 230 liters (Blanchard *et al*., 1987; Vandeplassche, 1987). Therefore, in most cases the fetus is not palpable, so the absence of positive fetal detection may be a suggestion to diagnosis. In the transabdominal ultrasound there is a large amount of fluid (>18 cm) including the non-pregnant horn (Zent and Pantaleon, 2008). The bilateral abdomen examination can exclude the possibility of twin pregnancy (Frazer *et al*., 2002). Ventral edema in the abdomen is usually present (Bucca, 2006).

Owners should be advised that this is a progressive disease and that is unlikely that the mare will maintain the pregnancy until term. Attempts to repeated transcervical drainage were reported, resulting in fetal death due to early placental detachment and/or bacterial contamination (Frazer *et al*., 2002). The parturition induction should be recommended before rupture of abdominal pre-pubic tendon or uterus happen (Hanson and Todhunter, 1986; Blanchard *et al*., 1987; Honnas *et al*., 1988).

The induction of parturition is a risk because hypovolemic shock and dystocia was observed in 50% of cases. The reproductive prognosis for the mare is good, when appropriate supportive therapies are instituted and provided no sequelae such as cervix laceration, retained placenta (Frazer *et al*., 2002) and metritis occur. According to Vanderplassche (1987), 6/8 mares that developed hydrops had normal pregnancy and delivery in the next pregnancy. Blanchard *et al*. (1987) reported one death in 2 mares due hypovolemic shock and abdominal muscles rupture with ventral inguinal hernia.

In hydrops cases the distended uterine muscle may not respond well to the oxytocin for parturition induction (Vandeplassche *et al*., 1972; Frazer *et al*., 1997b). Thus, the oxytocin and prostaglandin association can be used to induce abortion (Vandeplassche *et al*., 1972; Koterba *et al*., 1983; Waelchli and Ehrensperger, 1988). Prostaglandin E application in the cervix before induction may facilitate the fetus extraction. However a major complication is the swollen and thickened chorioallantois, which often must be mechanically ruptured (Vandeplassche *et al*., 1976). If finger pressure is not sufficient for the chorioallantois rupture, one can use a biopsy forceps to remove a membrane fragment and thus promote the liquid leakage (Vandeplassche *et al*., 1976; Waelchli and Ehrensperger, 1988). If required, a nasogastric tube may be inserted into the uterus to control the liquid siphoning (Frazer *et al*., 2002).

The intravenous fluid administration, concomitant with the gradual removal of allantoic fluid excess helps cardiovascular adaptation and oxytocin (1.0 IU/min) can be added to fluid to induce uterine contractions. The abdominal contractions can also be compromised due to the strain. Preventive treatment for laminitis-metritis complex is indicated mainly in retained placenta cases and uterine involution should be monitored by transretal palpation and ultrasonography (Frazer *et al*., 2002).

Ventral abdominal wall hernias and pre-pubic tendon rupture

Defects in the abdominal support for pregnant mares may result in disruption of transverse abdominal muscles, oblique, rectus abdominals and pre-pubic tendon, which attaches the pubis cranial border. The extreme distension in twin pregnancy, fetal membranes hydrops, trauma in late pregnancy and intramuscular or subcutaneous hematoma (Wilkins, 2006) may cause disruption of the ventral muscle-tendon support.

In extreme cases, rupture can lead to bleeding, shock and death (Ginther and Griffin, 1993). The most obvious clinical sign is the ventral edema in variable sizes that can extend from the most cranial region to the udder. Mares with tendon rupture of ventral pre-pubic abdominal wall show intermittent signs of colic and are often reluctant to move. If the tendon is torn, the pelvis is tilted, there will be lordosis and the mammary gland will be displaced cranium-ventrally because of the lost connection to the pelvis (Hanson and Todhunter, 1986). Unilateral edema is more indicative of damage to the ventrolateral abdominal wall, which according to Zent and Pantaleon (2008) seems to be more common in the mare, but can also occur in partial pre-pubic tendon ruptures.

The clinical signs of tachycardia and tachypnea with no change in body temperature are usually the result of extreme pain (Hanson and Todhunter, 1986). Definitive diagnosis may be complicated on late pregnancy, when a good evaluation by transrectal palpation due to extensive edema and crepitus ventral abdominal wall impeding the ventral abdomen ultrasound evaluation (Ginther and Griffin, 1993). Assessment of the exact lesion size will be possible only after the fetus and fluids are delivered and ventral edema decreases (Frazer *et al*., 2002). The mare may be too sensitive and resist palpation of the region. Monitoring via creatine kinase and aspartate aminotransferase enzymes may help determining the injury severity.

Pregnancy termination is the recommended treatment, however the prognosis is extremely reserved and the damage may prevent future pregnancy, and thus a more conservative treatment and support may be indicated in order to maintain fetal viability. The use of anti-inflammatory can relieve the discomfort associated with movement restriction. Stands for the abdomen can be adapted from tires, canvas and leather straps. Reducing the diet amount or combination of a mild laxative may help reduce the stress associated with (abdominal eutiraria) defecation (Ginther and Griffin, 1993). Assisted deliveries must be made in mares with ventral herniation or rupture, possibly because they present difficulties in abdominal contractions to expel the fetus and placental fluids (Hanson and Todhunter, 1986). The possibility of herniation and organs strangulation is a complication and should be evaluated and corrected by surgery (Hanson and Todhunter, 1986; Wilkins, 2006).

Cesarean section should be considered in cases that prioritize the foal over the mare due to a reserved prognosis, such as tendon ruptures in the pre-pubic region without the possibility of surgical resolution (Bucca, 2006). Owners should be advised that in some cases, because surgical treatment is needed another

pregnancy is not recommended (Hanson and Todhunter, 1986) and embryo transfer is a feasible alternative for reproductive purpose for these animals (Frazer *et al*., 2002).

Uterine torsion

Causes of uterine torsion in the mare are not well defined. For Van der Weijden (1996) and Frazer *et al*. (2002) uterine torsion in the mare occurs before the end of the pregnancy and, when precedes the delivery time, results in 5-10% of dystocia in horses (Vasey, 1993). It is likely that the vigorous movements of the foal in late pregnancy are one determining factor for uterine torsion (Frazer *et al*., 2002). The uterine rotation degree might be 180 to 540º (Bucca, 2006) and most commonly occurring clockwise (Vaughan, 1980) and cranial to the cervix (Lyle and Paccamonti, 2010).

The main clinical sign is abdominal pain, and when presented close to the expected delivery date may be confused with the delivery mechanism itself, and even with gastrointestinal disorders, when severe pain is detected (Pascoe *et al*., 1981; Perkins *et al*., 1996). A delay in definitive diagnosis increases the likelihood of fetal compromise (Frazer *et al*., 2002).

Rectal palpation is essential to diagnose uterine torsion. The examiner should perform a thorough examination of the major ligaments of the lumbar region to confirm diagnosis and determine the torsion direction and severity. If the torsion is present the two ligaments pass above the uterus, and the ligament on the side of the torsion tends to be more caudal and palpable as a vertical band, while the opposite ligament is pulled horizontally on the uterus before moving ventrally (Frazer *et al*., 2002). When there is a 360º constriction, examination may be hinder (Pascoe *et al*., 1981). In these cases the determination of the torsion direction may be difficult and the ultrasound test can help show the uterine wall thickening and distension caused by uterine vascular compression (Frazer *et al*., 2002) as well as identify a possible uterine rupture (Bucca, 2006). A uterine rupture can be a torsion complication in mares (Pascoe *et al*., 1981), especially in advanced pregnancies where the torsion diagnosis is not made and delivery is induced (Vaughan, 1980).

Several techniques have been employed for the uterine torsion correction in the mare. If the pregnancy is at term and the cervix dilated enough one can try manual torsion reduction through the introduction of the examiner's arm and ventrolateral manipulation of the fetus, when the pregnant uterus should be moved sideways until you can undo the bow and twist. Using this technique, Vandeplassche (1993) reported 80% resolution in term pregnancies. Another option requires the obstetric chains tied to fetal limbs to undo the twist carefully rotating the fetus in uterus, avoiding injury to the foal and the uterus (Ginther and Griffin, 1993).

When it comes to a preterm pregnant mare and the exact side of the torsion is known (Fraser *et al*., 1975) the examiner can employ the technique of rolling the mare (Bowen *et al*., 1976), with the drawback that in the last trimester of gestation this technique can lead to uterine rupture (Perkins *et al*., 1996) and may also generate changes in the digestive system, early placental detachment and fetal hypoxia due to the anesthetic protocol. Thus, some authors recommend ipsilateral flank laparotomy in the side torsion and manipulation of the pregnant uterus as an alternative treatment for these cases (Pascoe *et al*., 1981). When the twist direction is not diagnosed the incision should be performed on the left (LeBlanc *et al*., 2004). The presence of the live fetus greatly facilitates the procedure and in cases of advanced pregnancy often requires a second incision in the contralateral flank allowing two surgeons to manipulate the uterus (Perkins *et al*., 1996). This technique facilitates the correct inspection and positioning of the uterus (Frazer *et al*., 2002). However, it is important to remember that the uterus may be friable, so extra care on handling should be performed to prevent lacerations and uterine ruptures (Vaughan, 1980). If the fetus is not viable, the mare should abort it naturally after repositioning the uterus, but it is recommended to monitor delivery so that any dystocia by static fetal anomaly should be corrected (Bowen *et al*., 1976; Vandeplassche, 1987).

The ventral celiotomy is performed if uterine torsion provides significant impairment, either when other abdominal structures are involved or when the operator cannot perform a reduction through the flank. The decision for this procedure is based on the mare's condition, duration and degree of abdominal pain, accurate diagnosis and professional experience. Access via ventral midline allows quick resolution of the torsion, evaluation of the structures involved and complication (Frazer *et al*., 2002), however requires a general anesthetic procedure that may compromise the fetal viability (Fraser *et al*., 1975).

The prognosis in uterine torsion cases in mares depends on the degree of vascular compromise and the gestation period of occurrence. According to Chaney *et al*. (2007), the survival rate in cases of uterine torsion in pregnancies <320 days was 97% for the mare and 72% for foal, while in more advanced pregnancies was 65 and 32%, respectively, whereas for the positive future reproductive outcomes were 67%. In general, when the fetus is alive and circulatory compromise is not an issue the prognosis may be good for both mother and fetus (Frazer *et al*., 2002). In chronic cases where there is great uterine alteration, ovariohysterectomy and maintenance of the mare for non-reproductive purposes may be recommended.

Uterine rupture

A uterine rupture can involve the endometrium, the myometrium or all uterine layers and most commonly occurs in the dorsal uterine wall (McCoy and

Martin, 1985). It is a complication that may be associated with fetal membranes hydrops (Honnas *et al*., 1988), fetotomy, and excessive manipulation during dystocia, fetal static anomalies, uterine torsion, and treatment in postpartum period and even in cases of apparently normal births (Lofstedt, 1993; Rossdale, 2004).

Occasionally, the foal´s hoof can force the dorsal uterine wall during the expulsive effort on delivery which sometimes may be identified by rectal palpation or as irregular opening in the uterine surface (Hooper *et al*., 1993), and in some cases the intestine may protrude by lacerations in the vulva. Other sites likely to be injured are the ventral body cavity and the pelvic end of the gravid horn (Bucca, 2006).

Clinical signs depend on the uterine laceration degree, and in partial lesions blood cells diapedesis and contamination of the abdominal cavity can occur. Peritonitis can develop after necrotizing endometritis, but it is not necessary a full perforation of the uterus to cause peritonitis (Blanchard *et al*., 1983). In cases of total rupture, abdominal contamination is evident and there is an increase in the white blood cells count and severe septic peritonitis detectable from 24-48 h post-lesion (Dolente, 2004). Abdominocentesis in these cases may be important in diagnosis, particularly with mares that have a previous delivery history involving dystocia and fetotomy (McCoy and Martin, 1985) with abdominal pain signs, fever, lethargy and anorexia (Bucca, 2006). Although prepartum uterine rupture is extremely rare, it should be considered in mares that exhibit abdominal pain signs and depression. Mares with vaginal bleeding containing large discharge of blood and clots must also be carefully examined (Rossdale *et al*., 1991; Paccamonti, 2001).

The uterus must be examined immediately after fetus extraction in every obstetric exam (Adams-Brendemuhel and Pipers, 1987; Frazer *et al*., 1997a; Rossdale, 2004). The immediate diagnosis of rupture by means of rectal palpation can be difficult, placental evaluation may or may not be useful in locating the lesion (McGladdery *et al*., 1992) and uterine integrity assessment by vaginal palpation can be hampered by the difficulty of assessing the extremities of the uterine horns. Furthermore, the endometrial folds are large and edematous, which can also be a complication in the identification of uterine lacerations (Hooper *et al*., 1993). If a change is detected, the clinician must be extremely cautious when evaluating the laceration. A laceration that extends only to the myometrium can be easily converted into severe laceration due to an improper palpation (Frazer *et al*., 1997b). The visualization of the lesion extent via laparoscopy may be particularly important in determining the prognosis (Bucca *et al*., 2005).

The conservative clinical treatment aims to prevent circulatory shock and peritonitis (Ginther and Griffin, 1993). In small laceration cases, on the back of the uterus with little hemorrhage associated, the uterine therapy in the immediate postpartum period is contraindicated, however, there is a higher risk of developing a fatal peritonitis (Schimidt *et al*., 1991). This mare should receive systemic broad-spectrum antibiotic therapy, non-steroidal anti-inflammatory at an anti-endotoxemic dose (Perkins *et al*., 1991; Schimidt *et al*., 1991; Ginther and Griffin, 1993; Rossdale, 2004), and peritoneal lavage. Therapy with oxytocin can also be used to aid in uterine involution (Bucca, 2006).

Ventral celiotomy is recommended, especially for large lacerations, uncontrollable bleeding, viscera herniation, and when the mares do not respond to conservative medical treatment (Ginther and Griffin, 1993). Flank laparotomy can be performed in laceration cases of the uterine horn ipsilateral to the surgical incision (Rossdale, 2004). According to Hooper *et al*. (1993) the best treatment to save both life and the mare's reproductive potential is surgical repair of uterine rupture. The prognosis depends on the rupture size as well as the contamination degree. The earlier the treatment is initiated the better is the reproductive outcome (Rossdale, 2004). The survival rate can reach 90% in treated mares during the first 24 h after rupture (Schimidt *et al*., 1991).

Hemorrhage

Hemorrhage in the uterine wall and pelvic canal

When assisted delivery requires obstetric manipulation, slight bleeding may be evident from linear lacerations of the vaginal mucosal membrane, richly vascularized and edematous as a result of the physiological delivery preparative phase. This situation is complicated if implementation of traction force is necessary, especially in cases of bigger absolute or relative products (Prestes, 2006) and in nulliparous mares (Pascoe and Pascoe, 1988).

The hemorrhage may be profuse if injury occurs in the wall of major vulva veins, vestibule, vagina or cervix, resulting in localized hematomas (Prestes, 2006). In veterinary obstetrics the main severe hemorrhage cause is the laceration of blood vessels by instruments or obstetric maneuvers poorly performed (Noakes *et al*., 2001).

Most of the caudal reproductive tract is irrigated by the pudendal artery which branches in the vaginal artery (Ginther, 1992; Lofstedt, 1994). A hematoma in this vessel can stretch the pelvic cavity causing unilateral vulvar swelling (Rossdale, 1994). Broad-spectrum antibiotics and tetanus prophylaxis are indicated. Parts of the hematomas are resolved naturally, however some vulvar, vaginal or pelvic hematomas need to be drained within 7-10 days (Pascoe and Pascoe, 1988). It is important to differentiate vulvar hematoma from eversion or bladder prolapse (Asbury, 1993; Lofstedt, 1994). In some cases when bleeding is

confirmed through a structure, a hematoma can form and this may only be detected when the breeding pre-assessment in the first or second postpartum estrus is performed (Pascoe and Pascoe, 1988; Arnold *et al*., 2008).

In severe lacerations of the uterine wall, there may be leakage of blood vaginally, and in these cases, the mare should be confined in a stall and a low dose of oxytocin may be instituted. Uterine lavage is contraindicated because it may hinder the clots formation and prolong bleeding (Frazer *et al*., 1997a). Aggressive surgical procedures are avoided as the majority of cases resolve spontaneously (Frazer, 2002a).

Large vessels rupture

The hemorrhage due to large vessels rupture affects 2-3% of breeding mares (Arnold *et al*., 2008). Although bleeding may occur in the pre, trans and postpartum the highest incidence occurs in 24 h postpartum (Perkins and Frazer, 1994) and this is the most common cause of death in postpartum mares (40%; Dwyer and Harrison, 1993; Dolente, 2004).

The break in the mid third of the uterine artery is the most common occurrence (Rooney, 1964; Threlfall and Immegart, 1998), but occasionally may involve the ovarian or iliac artery (Arnold *et al*., 2008). Typically the break presents 2-3 cm long and is oriented parallel to the long axis of the vessel (Dwyer and Harrison, 1993). Generally, this break has a predilection for the right side due to left uterine displacement caused by the cecum; the uterine artery is more tensioned particularly in dystocia (Dwyer and Harrison, 1993). The bleeding can be directed to the abdominal cavity, restricted to the broad ligament, uterine wall, and uterine lumen or reach more than one of these spaces (Arnold *et al*., 2008).

The rupture incidences in large vessels increases with the number of births, mainly due to changes in elasticity and collagen fibers and are also related to the senile degeneration (Rooney, 1964). According to Grüninger *et al*. (1998) changes that affect the pluriparous mares resemble to what is called the “pregnancy sclerosis" in other species, with laceration of the internal elastic lamina, fibrosis and calcification of the endometrial vessels (Gruninger *et al*., 1998). However, according to Arnold *et al*. (2008) and coworkers in a retrospective study (1998 to 2005) 14% of mares with hemorrhage were young animals (5 to 7 yr) with a history of only one or two deliveries. Copper deficiencies in old mares are also related to the rupture of arteries in the peripartum (Dwyer and Harrison, 1993; Zent, 1997). According to Arnold *et al*. (2008) a previous episode of postpartum hemorrhage was considered a risk factor for only 5% of animals in the next pregnancy.

The urogenital hemorrhage diagnosis is based on clinical findings. The heart rate and the color of mucous membranes should be carefully observed in mares with dystocia (Frazer, 2002b). Some mares may show no hemorrhage sign as tachycardia, sweating, abdominal pain, depression, vocalization, and fasciculation (LeBlanc, 2008), while bleeding from hypertrophied vessels supplying the pregnant uterus can be fatal especially if the artery rupture occurs in the peritoneal cavity. In such cases the mare may be found dead or moribund (Perkins and Frazer, 1994; Zent, 1997; Sprayberry, 1999). If the bleeding is coming from the uterine broad ligament, mare can produce tremors and display signs of extreme pain (Dwyer and Harrison, 1993; Perkins and Frazer, 1994; Rossdale, 1994). These early signs may be confused with postpartum uterine contractions.

Sequential obstetrics exams should be performed in the animal whose deliveries need intervention (Dwyer and Harrison, 1993). However, this assessment may not be useful when the hematoma is small over the large amount of blood that is being or has been lost (Wilkins, 2006), in which case the transabdominal ultrasound examination and abdominocentesis can be of better assistance in detecting hemorrhage (Frazer, 2002b). The hematology cannot be changed early in the process due to splenic contraction, which temporarily keeps the hematocrit within normal parameters (Sprayberry, 1999). The transrectal assessment of the broad ligament may cause a sudden movement of the mare and this stress can cause a fatal rise in blood pressure so in cases of transrectal palpation in the postpartum period, the procedure must be done very carefully (Asbury, 1993; Perkins and Frazer, 1994).

The dilemma of how to treat a mare with hemorrhage from large vessels is deciding when to place a "bandage drug" and when the surgery is really necessary (Sprayberry, 1999). Exploratory surgery to repair the vessels rupture is contraindicated (Story, 2007). Moreover, scientific studies on hemorrhage in mares are still scarce and most recommendations are based on the collective knowledge of clinical trials and the methods used in humans. Implementation of the torn vessel ligation is usually not possible, but an artery forceps can be applied and left for 24 h. When pressure on the vessel cannot be done, one should hold vaginal compresses (Noakes *et al*., 2001).

In some cases the state of extreme hypotension may offer the animal a good chance of survival (conservative treatment), in other cases the restoration of intravascular pressure and circulatory volume should be attempted (Dwyer and Harrison, 1993; Sprayberry, 1999). Conservative treatment aims to reduce blood pressure, to form a clot at the site of rupture and restore cardiovascular volume (LeBlanc, 2008). Thus, it is recommended housing with limited space and calm environment, sedation, analgesia and no withdrawal of the foal due to anxiety that it can cause (Frazer, 2002b). The shock therapy requires a high volume of

intravenous fluid (2-3 liters of hypertonic solution followed by 20-30 liters of lactated ringer in 2-4 h or the hypertonic solution can be replaced by colloids (Sprayberry, 1999). Nasal oxygen therapy and blood transfusions are also indicated in serious bleeding cases. Non-steroidal anti-inflammatory can reduce inflammation and provide comfort to the mare (Dwyer and Harrison, 1993), and the administration of low doses of oxytocin help the uterine involution with uterine reduction in size and weight thus reducing the strain on the ligaments as well as reducing the amount of blood that reaches the uterine body (Rossdale, 1994). Some clinicians employ hypotensive resuscitation administering a vasodilator agent in conjunction with intravenous fluid. The idea at this point is to provide the blood volume maintenance when the animal has low pressure. Corticosteroids are indicated in cases of hemorrhagic shock, intestinal and pulmonary ischemia (Sprayberry, 1999). Therapy should be performed to protect the mare from multiple failures secondary to reperfusion injury and ischemia as well as preventing hematoma infection (Vivrette, 1997). In these cases, it is also necessary to prescribe anti-inflammatory and antioxidant drugs. Antifibrinolytic drugs may help in stabilizing the clot and pentoxifylline, which increases the red cells flexibility, can improve the oxygen support to ischemic tissues (Sprayberry, 1999).

The most common complications include fever, leukopenia, retained placenta, increased digital pulse, thrombophlebitis, and cardiac arrhythmias (Arnold *et al*., 2008). The prognosis in the acute phase is reserved. According to a study by Arnold *et al*. (2008) a higher survival rate than anticipated in the literature was obtained from mares that had antepartum and postpartum hemorrhage (60 *vs.* 87.3%, respectively) and 47% had normal birth again after recovering from the hemorrhage.

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