Proceedings des
Journées Annuelles de
l’Association Vétérinaire Equine Française

2 - 4 Décembre 2011 - Lyon, France

Next Meeting :
11 - 13 Oct. 2012 - Reims, France

Reprinted in IVIS with the Permission of the Meeting Organizers
PLACENTITIS: WHAT CAN WE DO?

STEFANIA BUCCA, DVM Somerton Equine Hospital, Friarstown, Kildare, Co Kildare, Ireland

Abstract

Placental pathology represents the most common cause of preterm delivery, fetal death, stillbirth and neonatal disease worldwide. Ascending placentitis is the most frequently diagnosed form of placental pathology and predisposing factors seem to play a major role in the pathogenesis of this condition. A large number of infective agents have been implicated, many being opportunistic or environmental invaders. Diagnosis of placentitis during gestation is often difficult as most mares show no outward signs of infection. Clinical signs usually indicate that long standing and often irreversible changes have occurred and they include premature mammary development/lactation and vaginal discharge. Mares with histories of placentitis on previous pregnancies should be investigated by serial ultrasound monitoring of the caudal utero-pacental unit and cervix. In utero stress due to infection appears to increase neonatal survival; however, these foals may have poor post-natal growth rates, incomplete ossification of the cuboidal bones and sepsis. Nursing care can be expensive and the foal may end up with a poor prognosis as an athlete.

Keywords: placentitis, cervix, mare, pregnancy, neonatal sepsis

Introduction

Placental pathology represents the most common cause of preterm delivery, fetal death, stillbirth and neonatal disease worldwide. Placentitis refers to inflammation of the chorioallantois, usually caused or complicated by infective agents and frequently extending to the amnion and the umbilical cord (funisitis). The inflammatory response may vary in intensity from slight to severe and show a variable distribution from localised to diffuse. In general, the location of placentitis reflects the route by which the infection entered the uterus. A large number of infective agents have been implicated, many being opportunistic or environmental invaders. Viral, bacterial, fungal and protozoal agents have being identified as causative agents of placentitis. Annual variations in numbers and types of placentitis reported in different geographical areas suggest the possible role of environmental factors in the incidence of the condition (i.e: MRLS in Kentucky 2001-2002).

Lesions Distribution

Ascending Placentitis.

Placentitis in mares is reported to be most commonly caused by microrganisms ascending through the vagina and breaching the cervical barrier. An active role of the cervix in the pathogenesis of the condition is therefore implicit. If there are bacteria and/or fungi on the cervix and the cervical seal is compromised, they can enter the uterus and cause placentitis. Anatomical, hormonal or even neurological factors contributing to cervical incompetence and/or increased perineal or vaginal contamination will increase the risk of ascending placentitis. Cervical inefficiency due to critical cervical shortening in mid gestation has been extensively described in women as a leading cause of preterm delivery.
with no convincing evidence as to the possible cause. Progesterone administration and cervical cerclage have been used as effective means to correct the condition in the absence of infection. A similar condition has been observed in pregnant mares in recent years and a similar therapeutic approach appears to be highly beneficial. Cervical placentitis tends to recur in some mares and preventive strategies should be implemented, including US monitoring of the cervix and cervical pole CTUP from mid gestation to term.

Ascending placentitis generally develops and progresses slowly during the course of weeks or months before clinical signs become apparent. The more chronic the infection the more extensive or pronounced the lesions of chorionic thickening and fetal growth restriction are likely to be. Depletion of chorionic villi, thickening, discoloration are commonly observed at the cervical pole of the affected placenta, often associated with a fibro-necrotic exudates. Thickening at the cervical star can prevent it rupturing at birth or abortion, so that the chorion tears across the rostral body. In about 12% of cases, infection will rapidly spread to the fetus causing septicaemia and abortion, before placentitis has become grossly evident. A variety of bacteria can be associated with ascending placentitis, but most commonly Streptococcus sp and E. Coli.

Diffuse or Multifocal Placentitis.
Less commonly diagnosed, is associated with haematogenous spread of microorganisms to the uterus of the mare with subsequent infection of the placenta. Occasionally mares may show pyrexia and signs of septicaemia prior to abortion. This form is usually associated with infection by microorganisms in the genera Salmonella, Histoplasma and Candida. In several countries an increasingly recognised example of abortion and stillbirth due to a diffuse placental villitis is that caused by a number of different leptospiral serovars.

Focal Mucoid Placentitis.
Also known as nocardioform placentitis, a chronic-active placentitis that occurs at the base of one or both uterine horns or rostral body. Gram-positive” nocardioform” filamentous branching microorganisms (Crossiella equi) have been associated with the distinctive lesions, where sites with villous loss are often coated in brownish, muddy, mucoid exudates. The infection causes late abortion, stillbirths or premature births and has emerged as the most commonly diagnosed type of placentitis during the last two foaling seasons. The pathogenesis of this form of placentitis is presently unknown, but it has been speculated the organism may enter the uterus at breeding time and remain at the most ventral aspects of the uterine body or horn throughout gestation.

Diagnosis
Diagnosis of placentitis during gestation is often difficult as most mares show no outward signs of infection. Some mares will show signs of premature mammary development and lactation, cervical softening and occasionally a vaginal discharge. Transrectal and transabdominal ultrasound examinations are very useful in diagnosing placentitis and assessing fetal viability. US features of a diseased placenta include diffuse echolucency (edema), increased CTUP (>15mm at term), loss of contact between uterus and chorioallantois, accumulation of fluid and/or echogenic material, cervical relaxation (prior to 9 months gestation). After delivery/birth gross examination and histopathology of fetal membranes will confirm US diagnosis.
Placentitis results in several outcomes. In addition to abortion, stillbirth and pre-term birth, the mare may produce small weak foals, small normal and normal foals. Small, weak neonates represent a special management and medical challenge, as they carry an increased risk of developing sepsis and orthopaedic problems and suffer a degree of prematurity. Small normal neonates usually result from mares displaying clinical signs for quite sometimes. These foals usually do well, as they have completed their fetal maturation stage in preparation of birth. They still carry a risk for sepsis and orthopaedic complications.

Treatment
Treatment of placentitis is often unrewarding, when the mare has developed clinical signs. Treatment aims at maintaining gestation for as long as possible to enhance neonatal viability, due to our limited ability at handling premature neonates. Every effort should be made to limit further infectious spread, reduce the inflammatory response and reduce the risk of increased myometrial contractility in response to inflammation. Experimental models examining the relationship between pro-inflammatory cytokines and placentitis showed that bacterial infection causes increased expression of pro-inflammatory cytokines leading to the release of PGE2 and PGF2α into the allantoic fluid and premature labor. Lyle et al. (2009) elegantly showed that the fetal hypothalamic-pituitary-adrenal axis is activated subsequent to infection or inflammation. Evidence suggesting that the synthesis of PGE2 and PGF2α is driven by cytokines in concert with fetal cortisol indicate that treatment strategies that fail to interrupt the inflammatory cascade and reduce fetal stress may be ineffective in preventing pre-term delivery.
References

- LEBLANC MM, GIGUERE S, BRAUER K, PACCAMONTI DL, HOROHOV DW, LESTER GD, O’DONNELL LJ, SHEERIN BR, PABLO L, RODGERSON DH. Premature delivery in ascending placentitis is associated with increased expression of placental cytokines and allantoic fluid prostaglandins E2 and F2 alfa. Theriogenology 2002;58:841-4