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Placentitis: What constitutes a mare at risk for late term abortion? How to approach these cases.

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Placentitis is the most important cause of premature delivery. Neonatal mortality results from birth of immature, underweight or septic neonates. Placental infections are often caused by opportunistic bacteria, with inflammation of the allantochorion causing premature fetal expulsion. Prevention of abortion depends on early detection, aggressive medical management to control infection, and minimization of proinflammatory stimuli to counter premature parturition.

The majority of affected mares are pluriparous. Many have caudal reproductive tract anatomical defects allowing introduction of pathogens to the cranial vagina and transfer to the uterine lumen via cervical incompetence. Placental infection and placentitis occur resulting in localized thickening of fetal membranes. Separation at the site of infection compromises nutrient transfer and gaseous exchange between the fetoplacental unit and dam. At the cervical star, thickening and separation of the allantochorion prevents rupture during delivery leading to fetal compromise. These areas may also act as reservoirs of infection for fetal sepsis.

Serial monitoring of at risk mares allowing early detection of placentitis is paramount in management. Mares that exhibit signs of placental compromise, have a history of placentitis or demonstrate systemic compromise should be monitored regularly starting the seventh month of gestation. Maternal signs are apparent after endocrine pathways are altered and inflammatory cascades are initiated. Presenting signs include precocious mammary development and vulvar discharge. Although vulvar discharge is prominent in experimental models, in clinical cases this may be scant, seen only with careful monitoring of the vulva, tail hairs and hind limbs. Transrectal ultrasonography of the caudal reproductive tract is commonly performed, allowing direct visualization of the cervical star region, fetal fluids, limited fetal parameters and combined thickness of the uterus and placenta. Expected values have been reported with deviations suggestive of placentitis. Transabdominal ultrasonography allows more complete evaluation of the fetus, allowing assessment of heart rate, activity and size. More widespread placental evaluation is achieved, with the ability to visualize lesions in the uterine body.

Three functions may be ascribed to endocrine hormones during pregnancy: maintenance of myometrial quiescence, stimulation of contractility during delivery, and promotion of fetal maturation. Hormonal changes in maternal plasma do not reflect early stages of fetoplacental pathology, rather changes are indicative of later stages of disease. Changes in maternal plasma progestin concentrations reflect placental abnormalities, with three progestin patterns observed. A premature rapid decline is seen when fetal death occurs or fetal expulsion is imminent. Placental pathology and fetal stress lead to a premature rise in progestins. A lack of the normal prepartum rise may also be seen where exposure to ergopeptine alkaloids from pasture endophyte fungus has occurred. Assessment of total plasma estrogens has been advocated as an indicator of fetal viability, however as the source is reported to be the fetal gonads this may not reflect health of the entire fetus.

To increase chance of success, therapy for placentitis must be initiated early. Treatment is directed towards curtailing microbial spread from the site of infection, maintenance of uterine quiescence and stopping production of proinflammatory cytokines which initiate...
an inflammatory cascade leading to premature fetal expulsion. A wide variety of antimicrobials are in clinical usage and their concentrations in allantoic fluid have been investigated. The use of progesterone or its analogues is widespread to quiet the myometrium and support histiotrophic nutrition. In addition to non-steroidal anti-inflammatory drugs, pentoxifylline to curtail proinflammatory cytokine production has been shown a rational treatment.

Successful management of placentitis is dependent upon early detection and prompt medical management. Control of infection and inflammation promotes uterine quiescence diminishing likelihood of premature fetal expulsion.