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Ascending placentitis is the most common cause of premature birth of foals and accounts for over 30% of premature births and foal losses within the first 24 h of life. Early diagnosis and treatment of affected mares is attempted because prolonging pregnancy may be associated with accelerated fetal maturation and delivery of a viable foal. Presently, methods for diagnosing impending preterm birth are limited in accuracy and treatment protocols are in their infancy.

Evaluation of mares suspected of having placental malfunction should include a physical examination, rectal ultrasonography, culture of vaginal fluids and in some cases measurement of plasma hormones and transabdominal ultrasonography. Management decisions, such as sending to a referral hospital and drug therapy, are based on clinical findings and the client’s ability to monitor the mare. Treatment regimens are designed to prolong pregnancy because chronic placental infections are associated with accelerated fetal maturation. Treated mares should be evaluated at least weekly to ensure that the fetus has not died in utero. The length of therapy and treatment intervals should be chosen carefully as fetal metabolism may differ from that of the dam.

Mares with ascending placentitis rarely exhibit systemic disease or abnormal blood parameters and are most frequently identified by the presence of a vaginal discharge or premature udder development. Of the 2 ultrasonographic techniques used in reproduction, transrectal ultrasonography of the placenta more accurately identifies mares with placental infection in late gestation than transabdominal ultrasonography because over 90% of placental infections are ascending. Parameters to be evaluated include placental attachment, placental thickening, clarity of fetal fluids and fetal movement. Weak fetuses tend to have limited mobility while uncompromised fetuses are active. Fetal fluids should contain sparse echogenic particles and no purulent material should be observed between the placenta and endometrium. The combined thickness of the uterus and placenta (CTUP) should be measured ventral and cranial to the cervix. The CTUP increases from a mean of 4.5 mm between 4 and 8 months to 9.5 mm at term. A CTUP greater than 12 mm after 9 months of gestation is associated with ascending placentitis.

Maternal signs of placentitis are manifested only after the disease process alters endocrine pathways and stimulates inflammatory and immune responses. Consequently, hormone patterns in maternal plasma often reflect later rather than early stages of a disease. Measurement of maternal progestin profiles and plasma oestrogen concentrations are of some use in identifying mares with placental malfunction. Total progestin concentrations in maternal plasma are low and do not fluctuate until 15–21 days before parturition when levels rise dramatically, only to fall precipitously 24 h before foaling. The prepardum rise in progestins is associated with development of the mammary gland and onset of mammary secretion electrolyte changes, whereas the decline is concurrent with an increase in fetal cortisol. Because progestins cross react with the progesterone antibody used in commercial radioimmunoassay and enzyme-linked immunosorbent assays, progestin concentrations can be measured in the maternal circulation in late gestation. Values may vary between laboratories because cross-reactivity differs between assays; however, progestin concentrations should remain constant with levels ranging from 2–12 ng/ml until the last 3 weeks of gestation. Three abnormal progestin patterns have been observed, a premature, rapid decline, a precocious increase or a failure to exhibit the normal prepardum rise. A rapid decline in progestins is most frequently seen in acute conditions where there is fetal death. A precocious rise in progestins is usually associated with placental pathology. A failure to rise in the last 3 weeks of gestation is almost exclusively found in mares exposed to ergopeptine alkaloids from the endophyte fungus found on tall fescue grass (fescue toxicosis). In general, mares with precociously high progestin concentrations are more likely to deliver live foals than those with low concentrations because
there has been some degree of fetal hypothalamo-pituitary-adrenal activity. A progestin profile can be determined by obtaining 3 samples of serum at 48–72 h intervals. Progestin concentrations that vary by >50% before Day 310 of gestation are associated with placental disease.

Measurement of a single plasma sample of total oestrogens also has been advocated as an indicator of fetal well being. A total oestrogen concentration >1000 ng/ml between 150 and 280 days of gestation is considered to be normal while concentrations <500 ng/ml have been associated with a severely compromised or dead fetus and levels between 500 and 800 ng/ml indicate a compromised fetus. It is doubtful that total oestrogen concentration can predict fetal death as the fetal gonads are unlikely to respond to fetal stress.

Treatment efforts are directed at combating infection, reducing inflammation and controlling myometrial activity. The majority of placental infections are caused by opportunistic bacteria such as *Streptococcus equi* ssp. *zooepidemicus*, *Escherichia coli*, *Pseudomonas aeruginosa* and *Klebsiella pneumoniae* migrating into the uterus from the caudal reproductive tract. Therefore, systemic antibiotics are given to affected mares. Of the antibiotics evaluated, only potassium penicillin and trimethoprim sulphamethoxazole reached therapeutic concentrations in allantoic fluid of mares with experimentally induced placentitis. Recent work shows that mares with experimentally induced placentitis delivered more live foals after treatment with trimethoprim sulphamethoxazole (30 mg/kg bwt, q. 12 h, *per os*), pentoxifylline (8.5 mg/kg bwt, q. 12 h, *per os*) and altrenogest (0.088 mg/kg bwt, q. 24 h, *per os*) than untreated infected mares.

FURTHER READING


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