Theriogenology Question of the Month

This feature is sponsored by the American College of Theriogenologists. Readers of the *JAVMA* are invited to submit contributions. Contributions should provide a learning exercise about theriogenology. A specific question should be posed for the readers. The author's answer to the question and a brief discussion should be presented. Possible topics include commonly seen problems in domestic or exotic animals. Herd problems in dairy and beef cattle, sheep, goats, horses, and exotic hoofstock, problems in kennels or catteries, or flock problems in domestic and exotic fowl also are appropriate. Please contact Dr. Craig A. Smith, Associate Editor (800/248-2862, ext 259, or FAX 847/925-1329), for further details.

History

A 14-year-old Quarter Horse mare was examined at 297 days of gestation by veterinarians in the equine field service unit of our institution to determine the cause of premature lactation. The mare had given birth to a healthy foal the previous year and was artificially inseminated 9 days after that foaling. Fourteen days later, a single embryonic vesicle was seen at the base of the right uterine horn during transrectal ultrasonography. Examination on day 36 of gestation confirmed a fetal heartbeat. The mare subsequently was vaccinated against **equine herpesvirus-1 (EHV-1)** at 5, 7, and 9 months of gestation.

Physical examination of the mare revealed that she was bright and alert. Rectal temperature was 37.8 C (100.1 F), respiratory rate was 18 breaths/min, heart rate was 48 **beats/min (bpm)**, mucous membranes were pink, and capillary refill time was < 2 seconds. A mucopurulent discharge was evident on the tail and surrounding perineum. Fetal movement was evident on per rectal palpation of the uterus. Combined thickness of the uterus and placenta was measured in the area immediately cranial to the cervix by transrectal ultrasonography,[1] using a 5-MHz linear-array transducer, and was found to be 7.5 mm (**Fig 1**). Transabdominal ultrasonography revealed a foal with continuous activity, which precluded obtaining a basal heart rate. Uteroplacental thickness was 1.6 cm; the measurement was obtained at the most dependent portion of the abdomen by use of a 5-MHz linear-array transducer.[2] Vaginoscopic examination revealed brown mucopurulent discharge on the floor of the vaginal vault. The cervix was not easily identified because of the position of the fetus. Using a sterile speculum, a sample of the discharge was obtained and submitted for aerobic bacterial culture. Mammary secretions were obtained by gently expressing one of the teats; 3 ml of fluid was obtained and a foaling prediction test[a] was performed in accordance with manufacturers' instructions. Results indicated the mammary secretions had a calcium carbonate concentration of 50 mg/dl.[3]

Treatment was initiated, using ceftiofur sodium (3 mg/kg [1.36 mg/lb] of body weight, IM, q 24 h) and progesterone in oil (1.1 mg/kg [0.5 mg/lb], IM, q 24 h). The mare was confined in a stall and placed on 24-hour observation. Two days later, the mare's condition had not improved, and she was admitted to our veterinary medical teaching hospital for further observation and treatment.

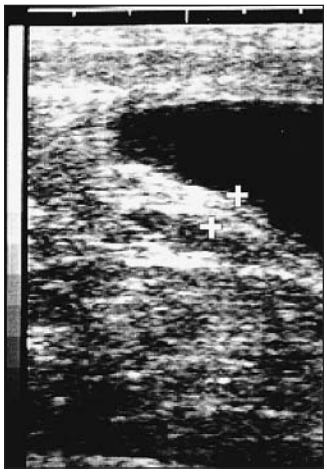

Figure 1—Transrectal ultrasonographic image of the uterine contents in a 14-year-old Quarter Horse mare with premature lactation on day 297 of gestation. Combined thickness of the uterus and placenta (distance between crosses) is 7.5 mm. The distance between each mark on the scale is 10 mm.

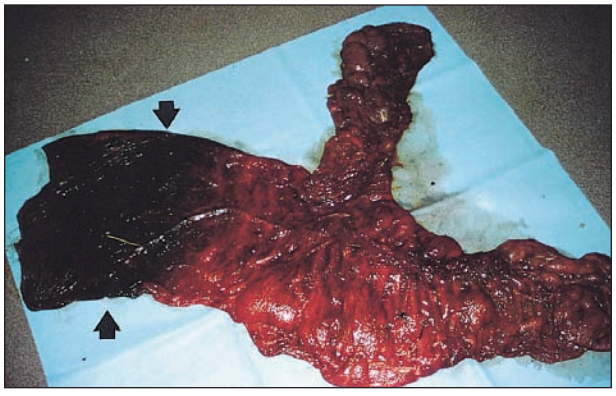

Figure 2—Gross view of the chorionic surface of the placenta from the mare in Figure 1. A specimen for histologic examination was obtained from the area of the uterine body (arrows).

On admission, the mare was quiet but alert. Transabdominal ultrasonography indicated a vigorous foal with a basal heart rate of 64 bpm (reference range, 60 to 92 bpm)[2]; other abnormalities were not detected. Other findings were similar to those recorded 2 days previously, and bacteria did not grow in aerobic culture. Treatment was similar to that started on the farm, but flunixin meglumine (1.1 mg/kg [0.5 mg/lb], IV, q 12 h) was added. Later that evening, the mare gave birth to a premature foal that weighed 30 kg (66 lb). The placenta was expelled, and it was examined for abnormalities. Lesions in the uterine body and the avillous area near the internal os of the cervix (ie, cervical star) were evident on gross examination of the chorioallantois (**Fig 2**).

Question

On the basis of the placental lesions, what is the most likely cause of premature lactation and parturition in this mare? Please turn the page.

Answer

Premature birth attributable to placental insufficiency that resulted from establishment of pregnancy in the uterine body with subsequent villous atrophy.

Results

In the mare reported here, there was evidence of a uterine body pregnancy with villous atrophy and mineralization of the placenta, especially evident during histologic examination (**Fig 3**). This mineralization may have accounted for the slight increase in placental thickness detected during ultrasonography and may have been attributable to severe chronic endometrial fibrosis near the cervical star.

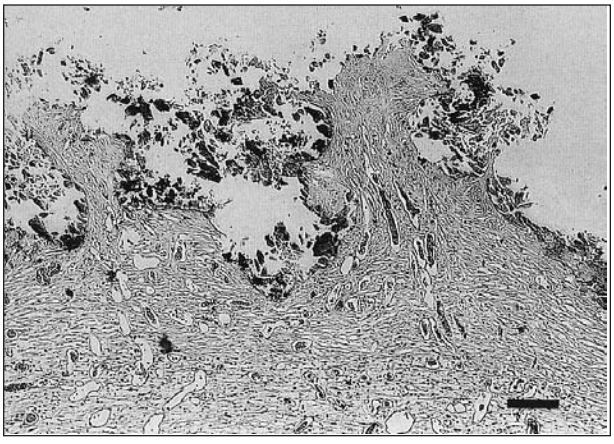

Figure 3—Photomicrograph of a section of placental tissue obtained from the area of the uterine body of the horse in Figure 1. Notice the villous atrophy and mineralization. H&E stain, 150 μm.

Discussion

Abortion in mares can be classified in 2 categories: noninfectious and infectious. The most common cause of noninfectious abortion in mares is twin fetuses, but ultrasonography has enabled veterinarians to better manage twins much earlier in gestation.[4] Other causes of noninfectious abortion include hormonal alterations, trauma, maternal stress, umbilical cord torsion, placental insufficiency resulting from degenerative uterine changes, hydrallantois, and pregnancy in the uterine body.[5] Most of the infectious causes of abortion include bacterial agents such as *Streptococcus* spp, *Escherichia coli*, *Klebsiella* spp, *Staphylococcus* spp, *Pseudomonas* spp, and *Leptospira* spp. Other agents include *Aspergillus fumigatus*, *Mucor* spp, *Candida albicans,* EHV-1, and equine viral arteritis.[4,6]

When treating a mare that has signs of impending abortion or premature parturition, it is important to isolate the mare. Fetal membranes should be promptly collected and the area where the mare aborted disinfected. Diagnostic tests should be used to rule out contagious causes of abortion. Historical information about the mare's pregnancy, clinical signs, environment, vaccinations, and recent transport or other stressors are important and may aid in diagnosis of the abortion. If the fetus is dead, necropsy should be performed to determine any gross lesions and to acquire organ and fluid samples for histologic examination, microbial culture, and serologic testing.

Another important organ for examination is the placenta. The placenta should be weighed to detect increase in weight attributable to possible edema and inflammation.[7] Characteristics of the placenta such as lack of villi, strictures and adhesions, thickening of the chorioallantois and amnion, and changes in color and texture may help to determine the causative factor.[4]

Placental insufficiency may result when the chorionic membrane is not in direct apposition with normal endometrium, thus resulting in improper villous formation.[4] In a normal placenta, 4 areas typically are devoid of villi: the cervical star, areas adjacent to endometrial cups, the uterotubal junction, and areas where the chorion folds on itself.[7] However, large areas of villous atrophy can lead to placental insufficiency and may predispose the mare to abortion or premature parturition because of inadequate nutrients for the fetus. Causes of placental insufficiency attributable to villous atrophy include twins, in which one chorion touches the other chorion, and chronic degenerative endometritis in which endometrial atrophy, cysts, or fibrosis interfere with placental formation.[5,8]

Typically, a pregnancy that develops in the uterine body causes a placenta that is underdeveloped in the uterine horns but has a large portion in the uterine body, as evident on gross placental examination.[4] The placenta barely extends into the uterine horns because of the growth of the fetus in the uterine body, and the placenta may be thick as a result of chronic inflammation.[4] Uterine body pregnancy is rare but is reported most often in older mares. The gestational length of these mares usually is shortened, with abortion as early as 7 months of gestation. Abortion typically results when the placenta cannot keep up with the nutritional demands of the fetus.[4]

The mare reported here was interesting in that, prior to foaling, she appeared to have placentitis. Combined thickness of the uterus and placenta and uteroplacental thickness determined during transabdominal ultrasonography were 7.5 mm and 1.6 cm, respectively. Typical placental thickness at day 300 of gestation is 4 to 7 mm (transrectal ultrasonography), and uteroplacental thickness during transabdominal ultrasonography is typically 1.26 ± 0.33 cm.[1,2] The slight increase in placental thickness (Fig 1) along with the abnormal vulvar discharge led to a tentative diagnosis of placentitis, although gross examination of the placenta revealed an enlarged uterine body and underdeveloped placenta in the area of the uterine horns, leading us to a presumptive diagnosis of uterine body pregnancy. Villous atrophy and mineralization that was detected arising from the area of the cervical star (Fig 2 and 3) led us to believe there also was secondary ascending placentitis. However, bacterial culture of the vulvar discharge did not yield growth after 3 days, and little evidence of inflammatory cell infiltrate was found during histologic examination of the placenta. After histologic examination, it was believed that villous atrophy was attributable to endometrial fibrosis in the region of the cervical star.

Mares with abnormal vulvar discharge must be

thoroughly examined to determine origin of the discharge. It may be difficult to distinguish between vaginitis and placentitis on vaginoscopic examination alone. Adding transabdominal and transrectal ultrasonography to assess fetal well being and placental thickness may aid in determining source of the discharge. Regardless of the source of the discharge, mares with evidence of premature lactation and vulvar discharge should be isolated and closely observed for premature parturition.

Treatment should consist of placing the mare on broad-spectrum antibiotics for up to 2 weeks, regardless of negative results on bacterial culture of vaginal or cervical contents. Antibiotic treatment may be altered on the basis of culture and susceptibility testing. Progesterone may be administered in an attempt to delay parturition, especially when the fetus is < 330 days old.[9] Nonsteroidal anti-inflammatory drugs such as flunixin meglumine (1.1 mg/kg, IM or IV, q 12 h) also may be considered to help minimize inflammation and reduce possible endotoxic effects on the fetus and dam. Although these treatments have not been documented for efficacy, it is our clinical impression that antibiotics and anti-inflammatory drugs are of benefit in horses such as this.

Outcome and Management

Failure of passive transfer was a concern because of the mare's premature lactation. The foal was given a transfusion with plasma shortly after birth, because specific gravity of the mare's colostrum was only 1.020[b] (target, ≥ 1.060). Broad-spectrum antibiotics also were administered. The premature foal reported here was evaluated to determine degree of cuboidal bone ossification, sepsis, and lung maturity; it was euthanatized because of evidence of septicemia and lack of cuboidal bone ossification. The mare did well and gave birth to a live, healthy foal the following year.

[a]FoalWatch, CHEMetrics Inc, Calverton, Va.
[b]Equine colostrometer, Lane Manufacturing, Denver, Colo.

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