Failure of pregnancy in horses and camelids

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Abstract

Determining pregnancy failures in horses and camelids is approached as a diagnostic investigation of an individual animal. It begins with the preliminary discussion around expectations of the investigation and apparently, diagnostic success rate is high in horses. However, diagnostic success in camelids is low. General approach from a pathology point of view is to identify several of noninfectious fetal and fetal membranes' lesions and then determine the potential infectious causes; agents include viruses, eubacteria, protozoa, and fungi. Bacterial and fungal infections are mostly ascending infections whereas viral and protozoal are systemic infections. When an infectious cause is excluded, the focus is directed on potential noninfectious causes and particularly those with detectible lesions. Maternal evaluation, specifically, via endometrial biopsy and examination of fetus and fetal membranes (including umbilical cord) in horses, normally provide an explanation for noninfectious failure of pregnancy in several circumstances. Special attention should be given to fetal thyroid gland, tracheal contents, and musculoskeletal system, particularly, the medulla of long bones. In horses, noninfectious failure of pregnancy with no lesions (idiopathic abortion) is observed in a small percentage of cases. In camelids, it is a common 'diagnosis.'

Keywords: Horses, camelids, pregnancy, abortion, stillbirth, embryonic mortality

Introduction

Equine theriogenology is focused on the production of a live foal. Infertility in horses is common and only ~ 67% of mares produce a live foal.[1] Number of mares becoming pregnant and producing a live foal is increasing;[2,3] however, failure of pregnancy (FOP) has been an important impediment to produce a live foal. In camelids, estimates are not readily available.

Investigations of FOP in horses is a most rewarding diagnostic experience because success rate is very high (83 - 93%),[4-7] higher than other species. Camelids, in contrast, like other species, have a success rate of up to 50%.[8] Major difference between horses and other species is the commonality of noninfectious causes in horses that can be identified by lesions visible at gross/macroscopic evaluation. This will be the focus in this review.

Failure of pregnancy in horses and camelids is described in detail in review articles, textbook chapters, symposium proceedings, and diagnostic laboratory pamphlets. Most diagnostic laboratories have a procedure, protocol, and/or list of diagnostic samples to aid in identifying infectious FOP in horses and to a lesser extent, in camelids. Most reviews and descriptions follow a familiar theme; separation of causes of FOP into infectious causes and everything else is based on the ease with which diagnostic laboratories can process samples to identify microbial agents.

In camelids, most FOP cases are considered idiopathic; they have no infectious causes and have no lesions. Many reports on causes of FOP are based on sample submissions to diagnostic laboratories.[4,5,6,9] This approach is logical and practical. Overlying this is the division of causes into those with identifiable lesions or those with no lesions. Fortunately, the number of cases in horses where there are neither infectious agents nor lesions identified (idiopathic FOP) is very low. Furthermore, causes are classified (based on origin) into maternal, fetal, or placental. Paternal causes possibility is also included in this review. The intention is not to replicate work already presented, but rather to highlight some important aspects and lesions that are easy to miss at macroscopic and microscopic evaluation. Additionally, 'idiopathic' failure of pregnancies is considered.

General approach to failure of pregnancy

There are 2 parts to the general approach to FOP. Preliminary discussions with owners to ensure safety, reasonable expectations, and thorough evaluation. Second, the diagnostic process.

Preliminary discussions begin before the diagnostic process and normally, includes discussing public health and safety issues, identifying the problem, performing a cost-benefit analysis of investigation and diagnosis, and discussing quarantine efforts of affected dams. In horses and camelids in North America, public health issues are minimal and in general, equine fetal membranes are not regarded as biohazardous above Level 2. Because horses and camelids are treated as

individuals, attempts for them to become pregnant and give birth are costly, there is usually a benefit for evaluation particularly, in horses where the diagnosis rate is so high. Highly infectious nature of equid alphaherpesvirus 1 (commonly known as equine herpesvirus 1; EHV-1) in horses means that every case should be evaluated for viral presence. This alone is a major focus in FOP investigation in horses even though the prevalence of EHV-1 in diagnostic material is relatively low and is ~ 3 or 4% in several large studies.[4-7,10,11]

Diagnostic process is like any other disease investigation and involves determining signalment, history, macroscopic evaluation, and collection of samples from the mare, fetus and fetal membranes, ancillary testing, and arriving at a diagnosis. Careful evaluation of fetus and fetal membranes and the collection of appropriate samples as outlined by a particular diagnostic laboratory must be carried out for complete evaluation.

During this process of sample collection, it is very important to have complete evaluation of all parts of fetal membranes and umbilical cord. When performing a postmortem examination on an equine fetus, evaluate thyroid gland, examine tracheal and bronchial contents, include skeletal muscle and sectioning of long bones. Reporting findings is now greatly enhanced by taking photographs with a mobile phone; this is sufficiently helpful when seeking opinions from diagnostic and or reproductive pathologists or equine theriogenologists. Careful macroscopical evaluation is very useful in identifying several noninfectious causes of FOP that in the horse represent 30 – 40% of overall causes.[4-7,10,11]

Infectious causes of failure of pregnancy

Infectious causes in horses account ~ 40% of the causes of FOP.[4-7,10,11]Stud owners give greater importance to equid alphaherpesvirus 1 because it is believed to have high infectiveness and has a propensity to move rapidly through a group of horses. However, it has not been a major infectious cause of FOP.[4-7,10,11]

Placentitis, either in the region of the 'cervical star' or in the body portion of fetal membranes (in nocardioform placentitis[12]), is a common lesion that when present is highly likely the cause of FOP; amnionitis is also a distinct lesion.

Approximately 70% of cases of placentitis have a microbe identified as the potential cause.[12,13] Majority of infectious causes of FOP are bacteria, and most common bacterial species are *Streptococcus equi zooepidemicus* or gram-negative organisms. These usually infect fetal membranes by ascending through the cervix. There are several potential bacteria that can cause FOP by the transcervical route. Other bacteria (e.g. nocardioform actinomycetes) are usually sporadic.[14]

Bacteria that infect fetus and fetal membranes include *Leptospira interrogans*, *Neorickettsia risticii*, *Borrelia burgdorferi*, *Chlamydia psittaci*, mycobacteria, and less commonly mycoplasma and *Coxiella burnetii*. Horses and camelids can be infected with these bacteria.

Fungal, protozoal, and viruses (other than equid alphaherpesvirus 1) are sporadic causes that can be recovered from diagnostic specimens with modern techniques particularly, molecular types (e.g. polymerase chain reaction-based assays).

Endometritis, including infectious endometritis, postbreeding endometritis, and postpartum endometritis all potentially contribute to FOP and infertility in mares. It results in embryonic mortality, particularly, early embryonic mortality.[15,16] Important components in the development of endometritis include variations in physical clearance and uterine tone, nonadaptive molecular immune response, and nonadaptive cellular immune response; these are areas of active research.

Uterine clearance and tone are especially important and systems to measure clearance and tone are being developed. Active inflammation of the endometrium is best detected by cytology and the effects of inflammation of the endometrium via endometrial biopsy. Endometrial biopsy continues to be an important method of prognosis for fertility in mares.[17-20] Evaluation of the mare is an important component of evaluating FOP; however, in middle or last third of pregnancy losses, the mare is often overlooked as a source of information about the fetomaternal interface and potential infectious agents. As with mares, camelids can be sampled for endometrial biopsy and this is a valuable adjunct to evaluation of infertility and FOP.

Amnionitis and funisitis is a unique presentation of infection in horses. Whereas a variety of bacteria can be recovered, the underlying process in the cause of disease, variously known as 'mare reproductive loss syndrome' or equine amnionitis, and fetal loss, was determined to be from the transmigration of setae of processionary caterpillars.[21-27]

Noninfectious causes of failure of pregnancy

Noninfectious causes of FOP represent a substantial component of the overall causes, ~ 50%. These causes can be classified into fetal, placental, maternal, fetomaternal incompatibility, and paternal components. Several of these have identifiable lesions whereas some lack. Noninfectious causes are classified into those with identifiable lesions and those without lesions. This aids in the investigation of the problem at the herd level.

Noninfectious causes with lesions

Noninfectious lesions of the mare, fetus, and fetal membranes are often identifiable at gross or macroscopic evaluation; however, some require careful evaluation of fetus and fetal membranes.

Noninfectious fetal lesions

Some lesions are highly visible congenital defects, and include hydrocephalus, craniofacial anomalies, schistosomus reflexus, and various musculoskeletal defects including arthrogryposis and angular limb deformities. Fetal neoplasia such as hepatoblastoma,[28,29] although extremely rare, is often very remarkable and do not present a challenge.

Dystocia or birth trauma have fetal abnormalities without routine histological changes and can only be determined at postmortem. Twinning once was a major cause of FOP in mares[10] but is now uncommon. It was diagnosed as a cause of FOP based on gross examination of the fetuses and fetal membranes.

Causes with a histologic lesion but no gross lesion requires collection of the appropriate sample. Approximately 3% of foals with glycogen branching enzyme deficiency are aborted and histological evaluation of muscle is required.[30] Thyroid hyperplasia observed with and without musculoskeletal disease, and thyroid hyperplasia/congenital hypothyroidism-dysmaturity syndrome requires microscopic evaluation of the thyroid gland for diagnosis. Thyroid histology must be combined with evaluation of long bones to detect osteopetrosis in those cases that do not have skeletal abnormalities such as mandibular prognathia, inappropriately ossified carpal and tarsal bones, flexural deformities of the forelimbs and ruptured tendons of the common digital extensor muscles.[31-34] Giant cell hepatopathy[35] is a lesion of unknown cause identified in aborted fetuses that have no other identifiable causes of FOP.

In camelids, anomalies of the head are the most common, especially choanal stenosis. Cardiac anomalies are also reported.

Noninfectious umbilical and fetal membranes' lesions

Examination of fetal membranes and umbilicus in equine FOP is a rewarding and reassuring experience once the investigator is familiar with normal fetal membranes and umbilical cord.[36, 37]

Macroscopic evaluation of fetal membranes and umbilical cord in horses should always begin with measurement of the cord for length, as excessively long or excessively short cords cause FOP. The length of cord varies among ponies, Thoroughbreds and Standardbreds, and draft breeds. For Thoroughbreds and Standardbreds there is some variation in what is considered the normal reference range and the general figures[38] of 36 - 83 cm is a good guide. Cords that are too short can potentially rupture or pull fetal membranes from the endometrium at the insertion of the cord.[39] Cords that are too long may have greater resistance to blood flow, become strangulated and wrap around a limb, the neck or the body, develop excessive torsion or develop an infarct of the placenta.[6,39,40] Excessive twists that are substantial may have evidence of torsion, particularly edema of the umbilical cord and dilation of the urachus. Other abnormalities of the umbilical cord include a single umbilical artery[9,41] strangulation(s) by the yolk sac remnant, a knot in the umbilical cord, umbilical hernia into the umbilical cord or the lesions of amnionitis and funisitis as occurs in mare reproductive loss syndrome/equine amnionitis and fetal loss. Camelids normally have a relatively long cord, and torsion and strangulation(s) occur sporadically.[42]

Ingestion by mares of processionary caterpillars begins as a noninfectious process however the setae of the caterpillars move through the intestines into the uterus and placenta carrying with them various types of microbes.[26,27] Limited information is available regarding pathogenesis of hydrallantois and hydramnios in mares for clinically obvious reasons.[43]

An abnormal site of attachment of the embryo to the uterus resulting in an umbilical cord attached to the body of the uterus results in body pregnancy and fetal loss. Formation of body pregnancy is an anomaly (limited information is known) and research in equine embryo attachment to the endometrium may provide insights in this area.[44]

Currently, placental edema and prolonged pregnancy following ingestion of mycotoxins as part of fescue endophyte toxicoses,[45,46] occurs sporadically.

Nutrition of the fetus and fetal membranes via endometrium occurs through a combination of histotrophic and hemotrophic means.[47] Secretions of endometrial glands are required for the uterine 'milk' that is taken up by trophoblasts at the base of the microcotyledons. Adequate microcotyledonary surface area is required for hemotrophic transfer. Endometrial disease, particularly, fibrosis and alteration of endometrial glands, is likely to alter placental development and the resulting reduced gland secretion, transfer of nutrients and surface area. Endometrial biopsy is the only effective way to measure alteration to endometrial structure that could influence placental surface area. Quantitative means to measure surface area in a diagnostic setting is not established. Endometrial biopsy therefore remains an important component of investigation of FOP and infertility in horses and camelids.

Twinning reduces the surface area of each placenta and is responsible for FOP. Twinning is now an unusual finding because of early detection and clinical intervention. It is more common in those animals that were not subjected to pregnancy testing. The prevalence of twinning is ~ 0.1% of pregnancies.[48]

Noninfectious causes without lesions

The most difficult group to identify as the cause of FOP are those that are noninfectious, and which do not have lesions identifiable either macroscopically or microscopically. This is the group that includes maternal causes, fetomaternal incompatibilities and those causes that have toxic, nutritional, genetic or molecular anomalies.

Maternal causes may be readily identifiable and as such, fetal and placental examination may be done on farm but not sent to diagnostic laboratories. Thus, maternal illness seldom is included in laboratory statistics. Colic is a recognized predisposing factor for FOP.[49-51] Age of mare,[10,52] failure of a previous pregnancy,[10] endometritis,[53] endometrial fibrosis,[18,54] uterine neoplasia,[55] and hormonal failure [2,56] are all reported.

It is generally considered that the majority of genetic or molecular causes of FOP are responsible for embryonic mortality and return to estrus. It is variously called repeat early embryonic loss, equine early embryonic loss or early pregnancy loss.[57,58] Cases lacking macroscopic or microscopic abnormalities have no known cause, partially because routine evaluation of chromosomal or molecular abnormalities is not available.

There are some reports of chromosomal abnormalities in the fetus including mosaicism [51] or chromosomal or molecular abnormalities in mares, including single nucleotide polymorphism[60] or chromosomal translocations.[61] This is a field where ongoing research is necessary.

Paternal causes of FOP have received limited attention. It has long been recognized that some stallions have an abortion rate that is higher than others[10] but there is limited research in this area.

Conclusion

Diagnosis of FOP, especially in the mare, is a rewarding process with a very high rate of diagnostic success. A search for noninfectious causes including placental and umbilical cord lesions, and evaluation for unique diseases in mares and fetuses, particularly, bone, muscle, and thyroid tissues, increases the diagnostic success rate even further.

Conflict of interest

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