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EQUINE DISEASE QUARTERLY

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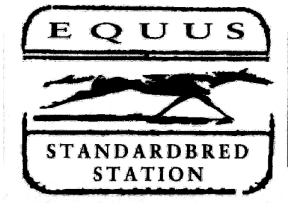

COMMENTARY

Investigating cases of toxicant exposure and why there is no single 'tox screen'

In cases of sudden death, abortion or neurologic diseases, a toxicant is often included in the list of possible causes of disease. If toxicants are suspected, a complete history and potential list of possible toxicants will help guide testing. No true 'comprehensive' toxicant screen exists in human or veterinary toxicology. Even in human toxicology laboratories, "routine" or "comprehensive" toxicology screens are generally limited to ethanol (alcohol) and therapeutic and illicit drugs and medications, which vary according to regional prevalence. Horses have a wider range of potential for exposure to toxicants, which varies based on their environment and management practices. When possible, a veterinary toxicologist should be consulted prior to sample submission to provide input on likely toxicants; advise on sample collection and handling; and assist with selection of appropriate analyses.

Clinical signs, physical examination findings and results of routine laboratory analyses (e.g., CBC, serum chemistry, urinalysis) in toxicoses are often nonspecific and indistinguishable from non-toxicologic causes of disease. A thorough and detailed history is therefore one of the most important tools in identifying potential toxicant exposures. For example, a history of multiple sick or dead animals with similar signs over a short period of time is one of the most common indications of potential toxicant involvement. Important details include recently opened bags of feed or new bales of hay; new medications or supplements; new water sources or interrupted access to water; recent movement to a new barn or pasture; recent weather changes or severe weather events; recent nearby landscaping, tree trimming, or gardening; recent nearby application of pesticides, fertilizers or other agricultural chemicals; and any other changes on or near the farm (e.g., construction, drilling or mining activity). Once potential toxicants are identified, analytical confirmation can be considered. Analytical techniques exist for some, but not all, toxicants. A veterinary toxicologist can direct testing to ensure the correct sample is chosen for the most appropriate test. In many cases, analysis can confirm exposure but data on relevant blood or tissue concentrations is lacking. Therefore, the combination of clinical signs and confirmation of exposure will lead to a presumed diagnosis of intoxication. Importantly, most analyses available at veterinary diagnostic laboratories require an idea of what class of toxicant is suspected. For example, a request to test for "rodenticides" or "rat poison" alone is insufficient to determine what analyses are appropriate. Several laboratories have a method that can measure warfarin, diphacinone, brodifacoum and other anticoagulant rodenticides.

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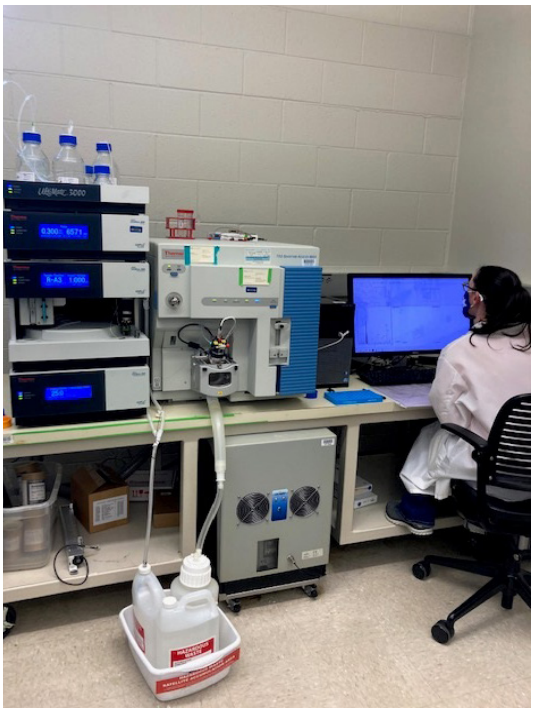
Analyses for other rodenticides, including the neurotoxicants bromethalin and strychnine, the vitamin D analog cholecalciferol and the metallophosphide zinc phosphide, however, each require entirely different tissues, sample preparation steps, reagents and instrument settings or entire instruments. The history, clinical signs and laboratory analyses can help indicate which toxicants are likely and should be tested for. Analytical methods are not available for every potential toxicant and so confirmation of exposure is not possible in every case.

In addition to choosing relevant analyses, sample selection and handling is key to successful toxicological testing. Some toxicants are distributed widely throughout the body and can be detected in multiple tissues. Others concentrate in a particular tissue and may be undetectable in others. Additionally, other toxicants are present in such small amounts in tissues that suspected source material (such as feed) is the best option for testing. For some toxicants, container selection is extremely important. For example, a water sample collected in a used sports drink bottle that contained electrolytes is not suitable for sodium quantitation. However well the bottle is rinsed, the previous substance will contaminate the collected water. Several resources are available to guide sample collection, handling, and shipping to facilitate accurate analytical results.

One example you find here Representative sampling is crucial to obtaining accurate results, particularly in contaminated feeds. Many toxicants in feed are not uniformly present throughout a bag or batch, but in localized "hot spots." Multiple smaller samples should be collected from different areas in the bag or bin, ideally with a grain probe. If a probe is not available, ½ to 1 cup samples should be obtained from various depths and locations within a container. If hay or alfalfa is implicated, bales or bags should be opened and examined carefully for weeds, leaves, flowers, berries, seeds or other unusual or unfamiliar plant material moldy, dusty or wet areas; blister beetles and other insects or insect parts; animal carcasses or fur; plastic,metal or other foreign material; and anything else unusual. With square bales or bagged forage, it is not uncommon for a contaminant to be present in only a few bales or bags, or even within a few flakes or cubes, while the rest is normal. While large round bales are more difficult to handle, they should still be examined as closely as possible. Areas of obvious contamination can be submitted for testing, or core samples can be obtained if bales appear normal.

Providing the veterinary toxicologist with complete case information about a potential toxicosis, including the animal's history, clinical signs, physical examination findings, routine laboratory results, and any other known information, especially prior to sample submission, can facilitate accurate diagnosis and prevent frustration due to inappropriate sample collection or test requests.
In the U.S., suspected problems with animal feeds, treats, medications or supplements should be reported to the Food and Drug Administration's Center for Veterinary Medicine. FDA CVM regulates these products and investigates problems including adverse effects, lack of efficacy, suspected contamination and other issues. Because there is no central mandatory reporting mechanism for toxic exposures in veterinary species, FDA CVM relies on voluntary reporting of problems. Anyone associated with a case, including veterinarians, technicians, owners, trainers and farm managers, can submit a report through an online portal here or by phone to an FDA Consumer Complaint Coordinator. A list of complaint coordinators by state can be found here Additional information on reporting problems, including what information to have, is here As part of a consumer complaint investigation, FDA CVM can help facilitate testing of suspect products through its network of accredited partner laboratories, the Veterinary Laboratory Investigation and Response Network.



Dr. Megan Romano
University of Kentucky Veterinary Diagnostic Laboratory
megan.romano@uky.edu

2022 Third & Fourth Quarters

International report on equine infectious diseases. Urgent: A multiple-state botulism outbreak among horses was reported in the United States of America with affected herds in Louisiana, Texas, Colorado and New Mexico. The source of the toxin (see also article on equine botulism in this EDQ issue) has been traced to alfalfa cubes produced by Colorado-based Manzanola Feeds and labeled as 'Top of the Rockies' Alfalfa Cubes. The product consists of 1-inch cubes, packaged in 50 lb/22.7 kg bags and labeled with the following codes: 111222, 111322, 111422, 111522 and 111622.

The following report was composed with information provided by the University of Kentucky Veterinary Diagnostic Laboratory and Equine Diagnostic Solutions, Inc.—both in Lexington, Kentucky; IDEXX Laboratories, Germany; the International Thoroughbred Breeders Federation; the International Collating Centre in Newmarket, United Kingdom; and by the American Association of Equine Practitioners' Equine Disease Communication Center. This report is retrospective and does not claim to be complete. However, it provides an indication of heightened activity of relevant contagious or environment-linked diseases among equids. To further improve this data, it is encouraged to report laboratory-confirmed diseases of Equidae to the ICC.

Strangles is still the most consistently reported disease from various regions across North America and Europe, including the British Isles. Reporting included laboratory confirmed farm outbreaks as well as samples submitted to various laboratories.

Few outbreaks of equine influenza virus were reported from regions of North America and from various parts of Europe during the third quarter. Outbreak numbers increased significantly during the fourth quarter compared to third quarter for both areas.

Equine herpes virus (EHV) -4 or -1 respiratory disease was reported infrequently in the USA and more often from operations in the UK, Ireland and continental Europe for both quarters.

During the third quarter, one EHV abortion was reported from Japan and three from Argentina. For the latter country, one would expect a pregnancy in the third trimester, which is the most vulnerable period for EHV-1 associated abortion. During the fourth quarter, three abortions were reported in Kentucky (USA) and one in continental Europe. Equid herpesvirus-associated myeloencephalopathy (EHM) follows a seasonal pattern with the majority of outbreaks associated with the cooler times of the year (first, second and fourth quarters in the Northern Hemisphere). Hence, only a few (four single case) outbreaks were reported from North America and one from the UK during the third quarter. In addition to seasonality, EHM outbreaks are extremely rare in the Southern Hemisphere. It is therefore noteworthy to report a single case of EHM at the San Isidro racecourse in Buenos Aires, Argentina. EHM outbreak frequencies changed as expected for the fourth quarter with 10 EHM outbreaks reported in North America, three outbreaks in continental Europe and one outbreak in the UK. Within Kentucky, a single case of EHM was detected in a barn at Churchill Downs racecourse in Louisville. Several other horses from the same barn were found shedding EHV-1; however, no further horse developed EHM. Noteworthy, during the fourth quarter, information of a (large scale) EHV-1 outbreak with EHM and abortions involving multiple herds was received from the Punjab region of India. However, this information is awaiting official reporting as diagnostic laboratory results have not yet been shared. A little more than 20 equine infectious anemia (EIA) virus positive animals (with rarely two animals simultaneously on a farm) were identified in North America. Cases were evenly distributed over the third and fourth quarters. There were also reports of EIA from Italy (three reports) and Hungary (one report). Increased vector activity during the third quarter was likely the cause for the relatively high case numbers of alphavirus encephalitis (exclusively Eastern equine encephalitis) reported in the USA and Mexico (one case). Flavivirus, mainly West Nile virus (WNV), activity was increased for distinct areas of Europe and Northern Africa. Countries that border the Mediterranean basin reported approximately 35 cases; Algeria, Greece and Tunisia each reported a single case of WNV. Most Mediterranean basin cases were reported from Italy. Germany reported nine confirmed cases from its central and nort heastern states. Miscellaneous: Contagious equine metritis (CEM): Mostly single cases on 14 premises in Germany and France tested positive for Taylorella equigenitalis, the causative agent of CEM.

Additionally, a single case of African horse sickness was reported from Sub-Saharan Africa (Nigeria); a case of rabies was reported in the USA; and a case of Hendra virus infection was reported from Australia.

Lutz Goehring
University of Kentucky
Gluck Equine Research Center
l.goehring@uky.edu

Maxwell H. Gluck
Equine Research Center
Lexington, Kentucky USA, 40546-0099
Telephone (859) 257-4757
Fax (859) 257-8542
gluck.ca.uky.edu

Botulism in Horses

Botulism is a neuromuscular disease characterized by flaccid paralysis that is caused by neurotoxins produced by the bacterium Clostridium botulinum. Horses are one of the most susceptible species, with both individual cases and group outbreaks reported. C. botulinum is a Gram-positive, spore forming, anaerobic bacterium. Spores are found in soil and aquatic sediments throughout most of the world, but the distribution of types vary based on environmental factors. Eight types of C. botulinum neurotoxin exist and are designated A, B, C1, C2, D, E, F and G. While all cause similar disease, there is variation in geographic distribution, potency and antigenicity. In North America, botulism in horses is most often caused by type B toxin and less often by toxin types A and C1.

Three forms of equine botulism are recognized. Intraintestinal toxicoinfectious botulism (shaker foal syndrome), develops in rapidly growing foals 1-2 months of age. This form develops following ingestion and overgrowth of C. botulinum in the gastrointestinal tract, typically caused by type B. Young foals have a less developed gastrointestinal microflora, which can permit spore activation, vegetative growth and subsequent neurotoxin production. Proliferation of C. botulinum type B in gastric ulcers, foci of hepatic necrosis (diseased livers), navel or lung abscesses and skin and muscle wounds have also been reported.

Forage poisoning, the most common form of intoxication in adult horses, follows ingestion of preformed neurotoxin in poorly preserved hay, haylage, silage or other feedstuffs. Silage and large, round, hay bales are most frequently implicated in botulism caused by neurotoxins type A and B. In horses, intoxication is most commonly associated with forage and almost never with commercial grain. Forage with a pH >4.5 is most associated with the growth of C. botulinum and toxin production. Type C botulism is associated with ingestion of feed or water contaminated with an infected rodent or small animal carcass.

Wound botulism, a less common form, results following infection of the umbilicus or a wound, such as a castration site. Regardless of type, C. botulinum neurotoxin acts primarily at the neuromuscular junction. The toxins are internalized at the nerve ending and through a series of steps inhibit the release of neurotransmitters needed for muscle contraction, which results in flaccid paralysis. Once bound, toxin neutralization is not possible and normal neurotransmitter release from the affected neurons will only return once new motor end plates have been generated, which may take 10-14 days.

Clinically, symmetrical flaccid paralysis is a consistent finding with the onset and rate of progression dependent on the amount of toxin that is absorbed. Initial clinical signs include difficulty eating with apparent excess salivation, weak eyelid tone, weak tail tone and exercise intolerance. Affected animals also spend increased amounts of time resting due to generalized muscle weakness, which is associated with tremors, carpal buckling and ataxia. Additionally, pharyngeal and tongue paralysis predisposes the horse to aspiration pneumonia and quidding (dropping) their food. Paralysis of the diaphragm and intercostal muscles can also result in an increased respiratory rate and decreased chest wall expansion. Severely affected animals ultimately die from respiratory paralysis and cardiac failure.

Botulism should be suspected in animals with flaccid paralysis displaying the aforementioned clinical signs. Botulinum toxin does not affect the spinal cord or brain but does affect the cranial nerves; thus, symmetrical cranial nerve deficits in an animal with normal mentation can help differentiate botulism from other disorders. A mouse bioassay can be used to obtain a definitive diagnosis by inoculating serum or gastrointestinal contents from an affected horse into a mouse. However, horses are extremely sensitive to the toxin and false negative test results are common; the mouse bioassay identifies botulism from fecal samples in only about 30% of adult horses with clinical disease. Detection of antibodies to botulinum toxin in a recovering unvaccinated horse also supports a diagnosis of botulism. However, antibody detection has limited treatment impact due to the time required for antibodies to develop Demonstration of spores in the intestine is not diagnostic, as they can be ingested and detected as contaminants. A quantitative real-time PCR (qPCR) for detection of the neurotoxin genes of C. botulinum types A, B and C, exists but is not currently available for commercial use. The combination of false negative tests and lack of PCR testing means that a diagnosis of equine botulism is typically based on clinical signs, known exposure to a feed or wound source and/or response to treatment. Immediate treatment with a polyvalent antitoxin prevents further binding of the toxin to presynaptic membranes. However, antitoxin cannot reactivate neuromuscular junctions that have already been affected. Thus, antitoxin administration may have little effect in severely affected animals. Generally, only one dose of antitoxin is needed and provides passive protection for up to two months.

Antibiotics should be administered if toxicoinfectious botulism is suspected or if there are secondary lesions such as aspiration pneumonia or decubital ulcers. Antibiotics that can cause neuromuscular blockade and possibly exacerbate clinical signs, such as aminoglycosides, should be avoided and neurostimulants, such as neostigmine, should not be used. Supportive care, including the provision of a deep bed and a quiet environment, is essential. Frequent turning of recumbent animals, nasogastric feeding and fluid support for animals with pharyngeal and lingual paralysis, frequent catheterization of the urinary bladder, the application of ophthalmic ointments and ventilatory support may be required.

A survival rate of 88% has been reported in foals with toxicoinfectious botulism that received intensive nursing care (including mechanical ventilation and botulism antitoxin). However, this type of treatment is not available in all areas and is quite expensive. Without aggressive supportive care, the mortality rate is high, with death usually occurring one to three days after the onset of clinical signs. The prognosis is variable in adult horses that have ingested preformed toxin (forage poisoning), depending on the amount of toxin absorbed and the severity of clinical signs. Mildly affected animals may recover with minimal treatment while severely affected animals that become recumbent have a poor prognosis. The mortality rate has been reported to be as high as 90% in recumbent adult horses, with death occurring within hours of the appearance of signs. In animals that survive, complete recovery is most common, although development of full muscular strength can take weeks to months. Persistent tongue weakness, not affecting the ability to eat, has been reported following recovery. A type B toxoid vaccine is available and should be used in areas in which type B botulism is common; vaccination is particularly important in areas where neonatal botulism occurs. Importantly, the type B vaccine only provides protection against type B toxin.

There is no cross protection against type C toxin and there is not a type C toxoid vaccine licensed for use in North America. If botulism is suspected to have been caused by ingestion of preformed toxin in feed, an alternate feed source should be provided while the origin of intoxication is investigated. Potentially contaminated feeds should be either safely disposed of or sent for confirmatory testing. Silage, haylage and other fermented feeds should be fed with caution to horses because of the risk of botulism. Regulations requiring the reporting of botulism vary by state and country (e.g., botulism is required to be reported to the Kentucky State Veterinarian). If you suspect an animal has ingested botulism from a contaminated food source, the U.S. Food and Drug Administration's Center for Veterinary Medicine or other regulatory body should be contacted.

Nathan M. Slovis DVM, Dipl. ACVIM,
CHTHagyard Equine Medical
InstituteLexington, KY 40511
nslovis@hagyard.com

Toxicologic causes of abortion and stillbirth in horses in North America

Abortion and stillbirth due to toxicant exposure is uncommon in horses in North America, although incidence in other geographic regions can vary. Toxicant-induced abortions can be due to a compound's effects on the placenta, on the fetus, on the mare or any combination thereof. Any toxicant that produces significant clinical disease and stress in a pregnant mare could potentially lead to abortion as a sequela. Very few toxicants cause primarily or exclusively abortions with few or no signs observed in the mare. In North America, two of the best-characterized toxicologic causes of abortion in horses are ergopeptine alkaloids and swainsonine.

Ergopeptine alkaloids are most familiar to those in the Southeastern United States in association with tall fescue grass. Certain strains of tall fescue (e.g., Kentucky-31) are infected with the ergopeptine-producing fungal endophyte Neotyphodium coenophialum. Endophytes live within the plant, between its cells and are not visible to the naked eye. N. coenophialum primarily produces ergovaline and its epimer ergovalinine; other ergopeptine alkaloids are produced in much smaller quantities. N. coenophialum can also infect and produce similar ergopeptine alkaloid profiles within perennial ryegrass (Lolium perenne).

Ergot fungus, Claviceps purpurea, infects the seed heads of a wide variety of grasses and small-grain forages. Most grasses can be affected, as can oats, wheat, barley and many other plants. "Ergotized" seed heads develop sclerotia – clusters of dark brown or black cylindrical fungal seeds or grains. Ergot fungus produces several different ergopeptine alkaloids, including ergocryptine, ergocornine, ergocristine and ergosine, often known collectively as ergot alkaloids. Ergopeptine alkaloids can exert a wide variety of effects in animals that ingest them, depending on animal species, reproductive status, dosage and time of year. Reproductive effects can include increased rates of embryonic death, prolonged luteal function and failure of normal udder development and lactation.

The most severe and dramatic effects occur in late-term pregnant mares – abortion, premature placental separation ("red bag"), overmature/dysmature foals and potentially catastrophic dystocia that can result in death of the mare as well as the foal. Ergopeptine alkaloids act via several different mechanisms, some of which affect the fetus, some the mare and others the placenta. Endocrine disruption occurs in both the mare and the fetus. In the mare, the alkaloids bind to dopamine receptors, causing a decrease in circulating prolactin. In the fetus, adrenocorticotropic hormone (ACTH) secretion is inhibited. Placental insufficiency and edema result from vasoconstrictive effects of the alkaloids. Placental fibrosis and mucoid degeneration of arteries can also result.

Swainsonine is also produced by fungal endophytes. In North America, several species of Astragalus and Oxytropis, known as locoweeds, are the most significant hosts of swainsonine-producing endophytes. Locoweeds are found primarily in rangelands in the Western United States. Slafractonia leguminicola (Rhizoctonia leguminicola), the fungus that causes "black patch" disease of red clover and other legumes, also produces swainsonine. In other geographical regions, plants containing swainsonine include Swainsona canescens, a legume native to Australia; Turbina cordata and several Ipomoea species in South America and Australia; I. carnea, found throughout the tropics worldwide; and Sida carpinifolia in Brazil. Swainsonine affects cellular metabolism by inhibiting mannosidases, causing accumulation of incompletely metabolized oligosaccharides and glycoproteins, cytoplasmic vacuolation and eventually vacuolar degeneration in multiple organs.

Most animal species are affected, although horses appear to be the most sensitive. Classic signs of swainsonine toxicosis in horses include aberrant behavior and other neurologic abnormalities, however, in pregnant mares swainsonine can cause embryonic or fetal death and abortion.

Any disease process causing anemia and/or methemoglobinemia in pregnant mares can result in fetal hypoxia and death; potential toxic agents include anticoagulant rodenticides and compounds that cause oxidative red blood cell damage. Brodifacoum toxicosis was reported to have caused abortion in a pregnant Arabian mare. Although the mare survived with hospitalization and aggressive treatment, she aborted the fetus. Red maple (Acer rubrum) toxicosis was suspected to have caused abortions in Percheron mares, but confirmatory testing was not performed. The mares were critically ill and ultimately died, but necropsy was not performed to definitively determine cause of death or abortion. Any disease process causing anemia and/or methemoglobinemia in pregnant mares can result in fetal hypoxia and death; potential toxic agents include anticoagulantrodenticides and compounds that cause oxidative red blood cell damage. Brodifacoum toxicosis was reported to have

caused abortion in a pregnant Arabian mare. Although the mare survived with hospitalization and aggressive treatment, she aborted the fetus. Red maple (Acer rubrum) toxicosis was suspected to have caused abortions in Percheron mares, but confirmatory testing was not performed. The mares were critically ill and ultimately died, but necropsy was not performed to definitively determine cause of death or abortion. Other toxicants that can cause methemoglobinemia and hemolytic anemia include red maple hybrids, silver maple and sugar maple; Pistacia species; Allium species (e.g., garlic, chives, and others); propylene glycol; and phenothiazines. Several therapeutic medications can cause abortions, including prostaglandin (often used for the purpose), oxytocin and glucocorticoids. Finally, any toxicant that causes significant systemic disease and stress to a pregnant mare has the potential to induce abortion. Examples include ionophores, cantharidin, antibiotic-associated colitis, and many others. Diagnosis of toxicologic causes of abortion relies on a history of exposure to significant quantities of a toxicant and ruling out other, more common, causes of abortion. Confirmatory testing is rarely possible due to a lack of analytical methods and lack of established clinically significant concentrations of a toxicant. In other cases, the time elapsed between exposure and abortion may preclude detection of a toxicant in blood or tissues. In cases of suspected toxicologic causes of abortion, a complete necropsy including microscopic evaluation of fetal and placental tissues should be performed, and a thorough history including any possible exposures should be provided to the pathologist to direct potential ancillary testing, including consultation with a toxicologist.

Dr. Megan Romano
University of Kentucky Veterinary Diagnostic Laboratory
megan.romano@uky.edu

Cervical pole necrosis of the equine placenta

Cervical pole necrosis (CPN), also known as placental necrosis or placental infarction, is a poorly understood disease of the allantochorion and an infrequent cause of non-infectious abortion in horses. The pathogenesis of this unique lesion is not definitively known, but the necrosis (dead tissue) has been theorized to occur either secondarily to placental detachment at the cervical star or by ischemic insult (lack of blood flow) related to hemodynamic abnormalities associated with long umbilical cords and vascular thrombosis. The University of Kentucky Veterinary Diagnostic Laboratory database was searched for cases of CPN to better characterize this distinctive entity.

Fifty-seven cases of CPN were diagnosed at the UKVDL from 2013 to 2022 (figure 1). Thoroughbreds were overrepresented (52 cases), but cases were also identified in Standardbreds (two cases), a Warmblood (one case) and in unidentified breeds (two cases). Clinical histories associated with the mares ranged from no signs of impending abortion or no history (33 cases), treated for placentitis (10 cases), premature udder development (nine cases), vaginal discharge (seven cases), placental separation identified by ultrasonography (two cases), a fluid filled fetal abdomen identified by ultrasonography (one case) and hydrops amnion (one case).

CPN resulted in abortion (44 cases), premature birth with subsequent neonatal death or euthanasia (seven cases), birth of a viable term foal (three cases) and premature birth of a weak viable foal (one case). Two cases, which included only the placental membranes for examination, didn't note the foal's outcome.

Gross necropsy findings, in all cases, consisted of a well demarcated, paper-thin region of tan to green placental necrosis associated with the cervical pole in close proximity to the cervical star (figure 2). Necrotic regions were bordered by a red and raised border that was distinctly evident on the allantoic surface. In addition to the cervical pole lesion, rare cases also exhibited similar regions of necrosis in the gravid and non-gravid allantochorionic horns. Size was not always noted, but when recorded, the necrotic regions ranged in size from 16 to 800 cm2. Umbilical cord lengths averaged 89.4 cm (case study range 37-160 cm; normal range 32-90 cm), and placental weights averaged 6.2 kg (range 1.8-12.7 kg). Fetuses were estimated to range from 180 days of gestation to term (average= 272), averaged 82 cm (range 47-109) from crown to rump and weighed 25.6 kg (range 5.5-69.1 kg) on average.

Additional diagnoses with CPN included fetal septicemia without significant inflammation (seven cases), bacterial placentitis (five cases), various fetal malformations (three cases), bacterial placentitis with fetal septicemia (one case), fetal bacterial pneumonia (one case), in utero meconium passage with aspiration (one case), premature placental separation (one case), umbilical cord torsion (one case) and one combined case of leptospirosis and hydrops amnion.

Based on UKVDL data, cervical pole necrosis is an idiopathic, non-infectious, placental disease that can result in abortion, premature birth, delivery of a weak foal or delivery of a viable foal. CPN routinely results in less than 2% of submitted abortions to the UKVDL. Cases in this retrospective study indicate that CPN occurs in both male and female fetuses, most commonly in mid to late gestation and in fetuses with both normal length and long umbilical cords. Cases of CPN were occasionally associated with secondary bacterial infections, which likely developed due to bacterial invasion through the devitalized allantochorion. Future studies are needed to better understand this unique cause of equine abortion.

Dr. Alan Loynachan
Veterinary Diagnostic Laboratory
University of Kentucky, Lexington, Kentucky
alan.loynachan@uky.edu

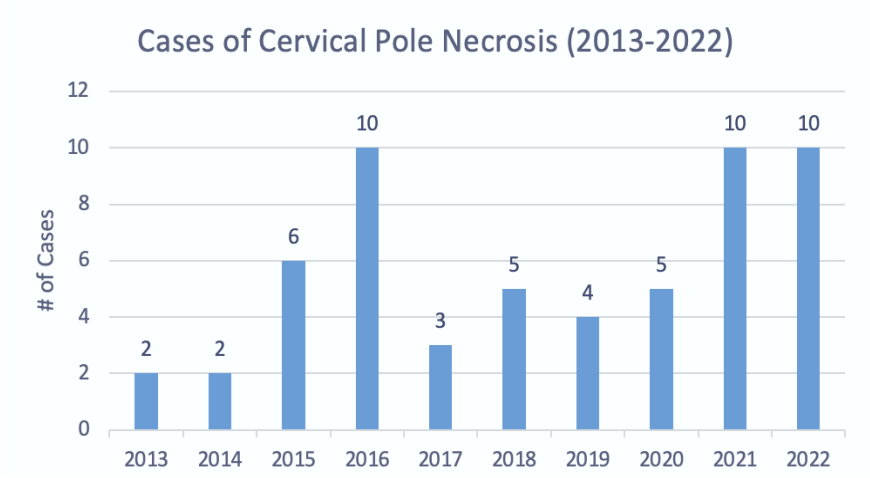


Figure 1. Cases of cervical pole necrosis diagnosed by the UKVDL by year (2013-2022).

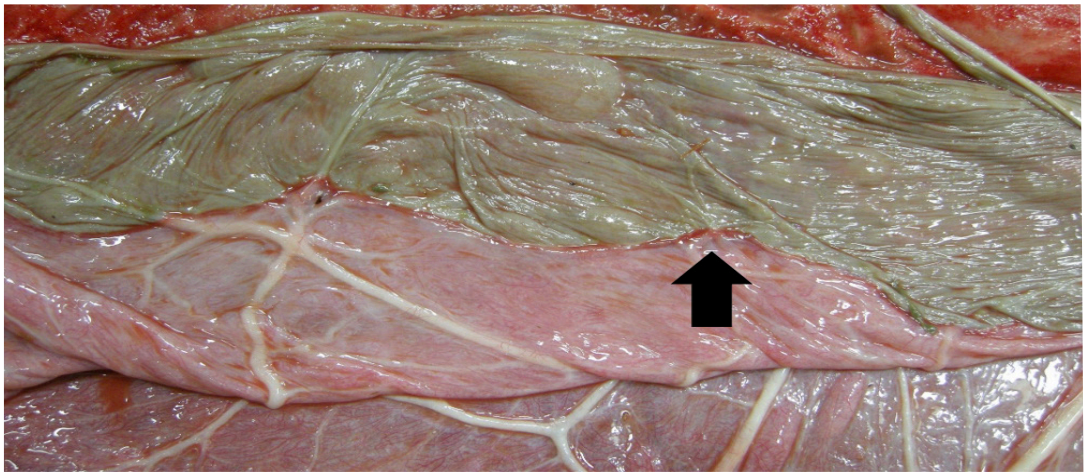


Figure 2. Allantoic surface of the allantochorion. A locally extensive and well demarcated region of the allantochorion is tannish-green and necrotic. A red, raised line (black arrow) separates the dead region of the placenta from the pink viable tissue.