SOME IMPORTANT DISEASES OF THE EQUINE NEUROLOGICAL SYSTEM

Jonathan H. Foreman, DVM, MS, Diplomate ACVIM
Professor, Equine Internal Medicine
University of Illinois
College of Veterinary Medicine
Urbana, IL 61802

INTRODUCTION

Neurological diseases can be some of the most devastating and dangerous clinical problems seen in horses. Due to their size, strength, and temperament, neurological horses can become a danger to themselves or to those around them.

EQUINE VIRAL ENCEPHALITIS (INCLUDING WEST NILE VIRUS)

Definition and history

Inflammation of the brain can be caused by a number of diseases, but one of the preventable causes is viral infection. In addition to Rabies, with which most people have some familiarity, two other families of virus can cause brain infections in horses, resulting in what is commonly termed viral encephalitis. The first and older group is the Alphavirus family (previously classified as Arboviruses, “arbo” meaning “arthropod-borne” or insect-borne viruses). In the USA these Alphavirus diseases include Eastern Equine Encephalomyelitis (EEE), Western equine encephalomyelitis (WEE), and Venezuelan equine encephalomyelitis (VEE, although not for decades in the USA). The other family which results in viral encephalitis in horses is the Flavivirus family, which includes West Nile Virus Encephalomyelitis (WNV).

It is believed that WNV was introduced into the USA in 1999 in exotic birds smuggled into New York. Over the subsequent summers between 2000 and 2004, WNV infections in horses and people gradually spread across the country from East to West until the virus could no longer be considered novel and the population of USA animals could no longer be considered naïve to the virus.

Epidemiology

Outbreaks of these viral infections occur as epizootics, or animal/human epidemics. For instance, in Illinois in 2002, over 1000 horses were infected and clinically diseased from WNV. Outbreaks occur in mid-to-late summer for two reasons. First, because that period provides the best mosquito breeding conditions (warm, wet) and second, because the virus “amplifies” in natural wild animal hosts before becoming sufficiently prevalent to result in domestic animal infections. In warmer climates (farther south in USA), the mosquito season may be quite long.

It is important to note that these viruses are no longer restricted to their original geographic distributions. While EEE tends to more eastern in the USA, Illinois is in the center of the area in the middle of the country where both EEE and WEE can be

prevalent. It is also critical to note that these viruses are all zoonotic, meaning that they have the ability to infect both domestic and wild animals as well as humans. It is, however, difficult for a human to catch one of these infections directly from a horse, because the horse tends to act as a "dead-end" host rather than as an amplifier of the disease like the wild animal natural hosts.

History may include an acute onset of CNS signs during the mosquito season, particularly if occurring in a multi-horse or herd outbreak. Infected horses usually are unvaccinated but spring vaccination may not be protective later in the year in an area where the mosquito season is particularly long.

Clinical signs
Non-specific clinical signs of viral encephalitis may include fever, anorexia, and rarely diarrhea (VEE). Neurological signs may include depression, dementia, aggressiveness, somnolence, and facial and other skin twitching/muscle fasiculations (especially WNV), followed possibly by blindness, recumbency, coma, and death.

Diagnosis
Diagnosis is based on history and clinical signs. Clinical pathology may evidence neutropenia and lymphopenia characteristic of a viral infection. CSF evaluation may document non-specific increased nucleated cell count (NCC: neutrophils, lymphocytes) and increased TP. More definitive diagnosis is based on plasma, serum, or CSF viral titers (Ames, Iowa) done with various standard viral techniques (HI, CF). WNV can be definitively diagnosed with plasma tested in more common venues with a capture ELISA for IgM (specific for natural WNV exposure, not vaccination). Definitive diagnosis is therefore based on a high initial titer; a 4-fold rise in paired titer; a positive capture ELISA for WNV IgM; or viral isolation (VI) from brain obtained fresh or frozen from necropsy. Other necropsy findings are characteristic but not pathognemonic of viral infection of the brain.

Treatment
Treatment consists of measures to control seizures as needed. Hydration must be maintained particularly if the horse is unable to eat and drink for itself. Antibiotics are sometimes administered to prevent secondary infections in horses which become recumbent. Treatment of decubital ulcers is also important in a recumbent horse, as may be the necessity to roll or sling the horse. While controversial, corticosteroids may improve WNV clinical signs (Illinois 2002, Florida 2001).

Prognosis
Prognosis is guarded because mortality is reportedly high: EEE 75-90%, WEE 19-50%, VEE 40-90%, and WNV 40%. Survivors may take several weeks to become normal or may always have some residual neurological deficits.

Prevention
Prevention is vital since mortality is high, treatment is non-specific, and yet good

vaccines are available. All horses should be vaccinated annually in the springtime (before the beginning of the vector season) and twice annually in areas where mosquito season is long (e.g. Gulf coast, southeastern coastal regions). Vaccines are available in various combinations including: EEE/WEE and EEE/WEE/WNV; and are often combined with other spring "shots" for tetanus, herpesvirus, and influenza.

EQUINE CERVICAL VERTEBRAL MALFORMATION

Definition and history

Equine Cervical Vertebral Malformation (CVM) is a developmental defect of the cervical vertebrae. As a result of abnormal growth, two adjoining vertebrae articulate abnormally, resulting in compression of the spinal cord as it traverses those two vertebrae. The most commonly affected joint is between the third and fourth cervical vertebrae (designated C3-C4).

These horses are often termed "wobblers" because they wobble or walk as if drunk or uncoordinated. Other synonyms for the condition include true wobblers, wobbles, Cervical Vertebral Instability (CVI), and Cervical Vertebral Stenosis (CVS). Many of these horses may be from families which typically produce large, fast-growing racehorses. These families may have a tendency to produce young horses with others signs characteristic of osteochondrosis (abnormal cartilage maturation into bone) such as a tendency toward rapid growth compared to their cohorts (age-matched pasturemates); osteochondritis dessicans (OCD) of the hock, shoulder, or stifle; and epiphysitis (asymmetry or overactivity in one or more of the growth plates of the long bones such as the distal radius at the level just above the carpus [knee] and the distal cannon bone just above the level of the fetlock).

Epidemiology

Because the disease is one of rapid growth and over-development, it is seen most often in young growing horses of large breeds. Yearling and two-year-old Thoroughbreds, warmbloods, and Thoroughbred crosses including appendix Quarter Horses are most commonly affected. Sucklings, weanlings and three-to-four-year-olds are less commonly affected. Older horses rarely develop CVM since most horses have achieved their adult height by four-to-five years old. Typically, affected horses have been overfed, with higher than necessary levels of dietary energy, protein, or both (Reed and Moore, 1993). Unfortunately, this rapid growth is encouraged in younger horses raised for the commercial weanling and yearling markets, where size and premature athletic phenotypes bring higher prices. Dietary imbalances in calcium/phosphorus ratio (1.5/1.0 recommended) and in trace mineral concentrations (copper and zinc) have also been implicated (Reed and Moore, 1993) but the data documenting their involvement are less clear.

Clinical signs

Signs usually begin gradually and insidiously. Early signs may be simply a mildly stiff neck and mild proprioceptive (limb placing) deficits. Gradually more obvious signs appear as

the spinal cord is compressed more severely. The affected horse may wobble more obviously, manifesting increased ataxia (loss of awareness of where its limbs are at rest and in motion), paresis or weakness (dragging toes, decreased range of motion), hypermetria (increased range of motion due to an inability to control movements in a normal range of motion), and spasticity (stiffness of movement, often observed as a decreased flexion of the hock and stifle joints). These signs are usually worse in the hind limbs than in the front limbs, due to the more superficial location and larger size (more susceptible to injury) of the hind limb nerve tracts in the spinal cord.

Neurological signs are graded on a scale from 0-to-4, where 0 is normal and 4 is falling down at normal gaits. A horse scored one grade worse behind than in front usually has a single, focal cervical lesion. Horses with the hind limbs graded two or more grades worse behind than in front usually have more than one lesion contributing to the neurological signs. Horses with hind and fore limbs graded equally may have a lower cervical lesion (e.g. C6-C7) resulting in some affectation of the lower motor neuron tracts to the fore limbs.

Diagnosis

Definitive diagnosis is made by obtaining cervical radiographs and a myelogram (Rantanen et al., 1981). Simple standing or recumbent cervical radiographs may be sufficient to demonstrate that there is so much narrowing of the spinal canal that the horse has CVM. If plain films do not definitively demonstrate a lesion, then a myelogram is indicated and is performed under general anesthesia. A radio-opaque contrast agent (sometimes incorrectly termed “a dye”) is injected into the foramen magnum, the opening between the base of the skull and the first cervical vertebra. The needle is placed deeply into the space between the overlying membranes (the meninges) and the spinal cord. Subsequent radiographs document any narrowing of the spinal canal by compression of the dye column. Flexion or extension of the neck (stressed views) may be necessary under anesthesia to re-create the condition in which the cord may become pinched as the awake horse normally moves its neck around while eating, drinking, and exercising.

Cerebrospinal fluid (CSF) is obtained before the contrast agent is injected during the myelogram. If the myelogram results are positive for CVM, the CSF may be saved but not analyzed. However, if the myelogram is negative, or if money is no object in diagnosing all possible causes of the horse’s ataxia, then the CSF is analyzed subsequently for evidence of Equine Protozoal Myelitis (described below), Equine Herpesvirus-1 Myelitis, meningitis, and neoplasia.

Treatment

Treatment of horses with CVM is controversial. Years ago these horses were all euthanized as being unsalvageable. Research and clinical experience at some universities since then has resulted in the development of various surgeries for stabilization of the pinched vertebrae and spinal cord (Wagner et al., 1981). The most commonly-performed procedure is ventral body fusion, in which a bone plug graft or,

more recently, a metal basket is placed into a pre-drilled hole to prevent flexion of the two vertebrae which make up the affected space. Over time the basket fills with new bone growing in from the sides, and the space becomes fused and unable to flex. Dorsal slot laminectomy also has been used to relieve spinal cord pressure in horses with static cervical vertebral stenosis diagnosed by myelogram without requiring flexion or extension to observe the lesion. The equine mortality insurance industry has embraced these surgical procedures and sometimes even requires that they be performed on insured horses under threat of policy vitiation.

Observed sequelae include cervical muscle atrophy either due to nerve damage or from disuse due to bone pain subsequent to surgery; fracture of one or both vertebrae; misplacement of the basket implant resulting in failure to stabilize the joint; development of instability in the joint rostral or caudal to the stabilized joint; and the whole litany of possible complications of general anesthesia in any horse including recovery fractures, pneumonia, diarrhea, and renal disease.

Another controversial method of minimizing neurological signs in these horses is the use of a nearly-starvation diet and stall confinement to reduce growth rate and minimize further development of osteochondrosis (Donawick et al., 1989, 1993). The premise is that by decreasing nutritional impetus for fast growth, the growth rate can be slowed and the already abnormal vertebrae can be afforded time to remodel until no cervical compression is apparent. This technique has been described twice in unrefereed literature but has not gained wide acceptance due to its possible humane aspects and due to its not having been evaluated in a controlled experimental setting. Young Thoroughbred horses were observed with very early, mild neurological deficits (Donawick et al., 1989, 1993). They then were fed poor quality grass hay, no grain, and no pasture, and they received strict stall rest over several months' time. The result was that most eventually had minimal neurological deficits and most went on to perform adequately as racehorses. Their marketablility was diminished by severely slowing their growth rate but their neurological signs were minimized without surgical intervention. A recent study by Hoffman and Clark (2013) showed that so-called "conservative management" resulted in 21 of 70 Thoroughbred horses treated medically, without surgery, raced (of the total of 103 horses in the study, another 16 were lost to follow-up and 33 were euthanized before reaching racing age). Statistically the more neurologically-severe horses tended to be euthanized and the milder-affected horses were those that went on to race without surgery.

Prognosis

Many horses which survive the immediate post-operative period improve as a result of the ventral fusion surgery. However, few if any are normal afterward; there are nearly always residual neurological deficits. From a liability standpoint, one must question the wisdom of having these horses, previously diagnosed as neurological, ridden and raced in the company of other horses.

Prevention
Preventive measures primarily involve careful breeding and feeding programs with slower dietary pushing for fast growth. Breedings observed to produce wobblers in the past should be avoided. Mares which have produced wobblers previously should be monitored carefully for level of milk production, as heavily-milking mares may be predisposed to producing faster-growing foals. This phenomenon has been most apparent to this observer in Thoroughbred foals raised on nurse mares of draft heritage. Creep feeding must be done judiciously to prevent overfeeding by a greedy suckling or weanling which pushes its pasturemates away from the feeder. Feeding for the commercial market may be necessary but again must be done judiciously to prevent creation of a large, well-muscled but ataxic yearling. Ideally allowing individuals to mature at a more natural rate of growth results in adults of similar size, although admittedly some may develop as athletes later than if pushed earlier.

EQUINE PROTOZOAL MYELOENCEPHALITIS

Definition and history
Equine Protozoal Myeloencephalitis (EPM) is a sporadic and sometimes fatal neurological disease of horses (but not donkeys or mules) caused by *Sarcocystis neurona*, a protozoan parasite which invades the horse's central nervous system. EPM in one horse is not contagious to other horses because the parasite stages in the infected horse do not produce protozoal stages which are infective to other horses. Synonyms for EPM include Equine Protozoal Myeloencephalopathy, Equine Protozoal Myelitis, Equine Protozoal Encephalitis, EPM, and "Protozoal," as in, "My horse has 'Protozoal.'"

EPM was first reported as a clinical disease in horses in Kentucky in 1970 (Rooney et al., 1970). In 1974, an unidentified protozoal parasite was first associated with spinal cord disease in horses (Cusick et al., 1974). Initially the organism was thought to be *Toxoplasma gondii* (Cusick et al., 1974) but was later shown to be a *Sarcocystis* species (Simpson and Mayhew, 1980). *Sarcocystis neurona* was first isolated from infected equine spinal cord and identified as the causative agent of EPM in 1991 (Dubey et al., 1991). In 1996, another protozoal parasite, *Neospora*, was also found to be associated with abnormal clinical signs of spinal cord disease in horses in California (Marsh et al., 1996).

Life cycle
Sarcocystis neurona has a classic two-host predator-prey life cycle similar to all other *Sarcocystis* species. The opossum (*Didelphis virginiana*) is the definitive host (Fenger and Granstrom, 1995). The opossum excretes the parasite in its feces. Intermediate hosts include: domestic cats (*Felis domesticus*, Dubey et al., 2000), raccoons, skunks, and nine-banded armadillos. In the intermediate host (the prey), the organism lives in the muscles. Opossums (the predators) eat the muscle of these intermediate hosts. The organism then escapes from the eaten muscle and sets up new life cycle stages in the intestine of the opossum. The definitive host (the opossum) excretes more eggs, and the parasite is then ingested by another intermediate host and the life cycle continues in a

circular or cyclical manner.

If a horse comes in contact with infective opossum feces (primarily on pasture or in hay where the opossum has taken up residence in the hay storage area), the horse may ingest the parasite and become infected. Once ingested by the horse, the parasite reproduces in the lining of most blood vessels and potentially can spread via blood (parasitemia) throughout the horse's body, including the central nervous system. Usually the parasite does not enter nervous tissue, so most horse infections are inapparent or subclinical. Recent data have shown as many as 50% of clinically-normal horses are seropositive for EPM (blood test positive), indicating exposure to the parasite, but only a small fraction of all horses actually develop clinical neurological signs (MacKay 1997; Saville et al., 1997; Bentz et al., 1997; Blythe et al., 1997).

Because the stages of the parasite in the horse are not capable of sexual reproduction, the horse is a dead-end host which cannot transmit the infection any further. An infected horse residing next to a normal horse is not contagious or dangerous to the normal horse. However, if the two horses have been living under the same conditions for several months, the normal horse is subject to the same routes of parasite ingestion which proved infective for the sick horse.

Epidemiology

In several epidemiological studies, approximately half of the clinically-normal horses tested were EPM seropositive in Pennsylvania (45% positive), Ohio (53% positive), and Oregon (45% positive) (Saville et al., 1997; Bentz et al., 1997; Blythe et al., 1997). Older horses were routinely positive more often than younger horses. These data illustrate that as horses age, they are more likely to have been exposed to the EPM organism at some point in their lifetimes. There were no significant breed or gender effects on seroprevalence. Seroprevalence was lower in areas with low rainfall (Blythe et al., 1997) and in areas with longer, colder winters (Saville et al., 1997). These findings imply that the organism cannot survive as easily in very hot and dry climates or in much colder (frequently below freezing) climates.

Except for the rare USA-exported horse which contracts EPM after leaving the country, EPM is restricted to those areas of the world where the host opossum may be found naturally. Clinical disease has been reported widely in North America (Fayer et al., 1990) and parts of Central and South America. Most cases are sporadic and isolated (singular) on a given farm, but widespread farm outbreaks have been reported occasionally in Kentucky, Ohio, Indiana, Michigan, and Florida. In the first report of a large number (n=364) of microscopically-confirmed cases of EPM, infection was most common in Thoroughbreds, Standardbreds, and Quarter Horses (Fayer et al., 1990). The ages of affected horses ranged from 2 months to >19 years old.

Epidemiological investigations have described several risk factors for the development of clinical EPM (Saville et al., 2000a). Horses had increased risk for developing EPM in spring, summer, and fall when compared to winter; when all feed materials (hay and

grain) were not secured from wildlife; when hay alone was not secured; when opossums had been observed on the farm; when there was a previous diagnosis of EPM on the farm; when horses were used as racehorses or show horses as compared to breeding or pleasure horses; and when horses had been ill in the 90 days prior to admission for EPM diagnosis. Horses had decreased risk for developing EPM when feed was protected from wildlife (opossums and birds) and when the barn or pasture was close to a creek or river. The proximity of creek or river bottoms probably provides a more attractive habitat for opossums which then have no need to invade the artificial environment of the barn and its feed sources. The implications from these data are obvious: whenever possible, horses must be kept in an opossum-free environment and feed materials must be secured.

Clinical signs

Clinical signs of disease may not occur for a year or more after ingestion of the organism. Stress (exercise, transport, pregnancy, illness) may precipitate signs in "carriers" of the organism. Fever has not been reported as a clinical sign. Neurological signs may present rapidly (acutely) or slowly and gradually (chronically). Acute onset signs are often rapid, severe, and debilitating, rendering the horse very ill. Chronic onset of disease means that the signs are usually slower and less severe in onset. A slower, more insidious onset of disease often carries a better prognosis since the signs may be recognized while still mild and treatment may be begun before the horse becomes very ill.

Most horses present with signs of spinal cord abnormalities. These signs include incoordination, stiffness, and weakness: the horse may walk "like it is drunk." These horses may be confused with true wobblers. More chronically, horses with EPM may develop muscle atrophy because of the decreased trophic input from abnormal spinal cord neurons. Especially when muscle atrophy is unilateral, EPM should be suspected since there is rarely a reason for other neurological diseases such as CVM or Equine Degenerative Myelopathy (EDM) to be lateralizing.

Occasionally, horses present with signs of brain disease, with severe intracranial signs including depression, blindness, walking in circles, and even an inability to stand. These horses usually deteriorate quickly and have a poorer prognosis. The severity of their signs means that they should be considered dangerous, and clients are encouraged to handle them with caution.

Sometimes signs may be very mild and may even be confused with lameness (Foreman et al., 1990). With these neurological-origin lamenesses, the source of the lameness cannot be determined through routine diagnostic testing such as nerve blocks, radiographs, and scintigraphs. There may be a history of stifle "locking." Some performance horses are reported not to "bend" as well as previously, or to misbehave at odd times (perhaps an early sign of brain disease).

Diagnosis

Diagnosis of EPM may be presumed from the history and neurological signs, especially if lateralizing. EPM testing may be performed on blood samples, but there is a 50% chance that even a normal horse will test positive on an EPM blood test. One EPM scientist has stated that "positive results of a serum immunoblot test have no value in ruling in a diagnosis of EPM" (MacKay, 1997). Practitioners often use serum screening as a method to rule out EPM as a possible diagnosis since EPM is unlikely to be the cause of the neurological signs if the serum is negative for EPM-exposure.

CSF is tested for EPM in three ways: a Western blot or immunoblot test for EPM antibodies (proteins which fight the EPM organism); an indirect fluorescent antibody test (IFAT); or a direct test for organism surface antigens (SAG 2,3,4). There is a high correlation (>90% agreement) between a positive immunoblot spinal fluid test and the presence of the parasite at post-mortem (Granstrom and Saville, 1998). There is a similarly-high correlation between a negative CSF test and a lack of organism at post-mortem. There is a poor correlation between being seropositive and finding the organism or its associated changes at post-mortem (MacKay, 1997), although very high titers (>1:4000) on SAG 2,3,4 tests are believed by some to be indicative of recent exposure and therefore true EPM.

Treatment

The original method of treatment of EPM was oral administration of drugs classified as folic acid inhibitors (sulfonamides and pyrimethamine) for a minimum of 90-120 days. These drugs prevent folic acid production within the protozoa, resulting in their death. They usually do not cause folic acid deficiency in the horse since horses absorb considerable dietary folic acid from their intestine as long as they are eating a good quality diet with either grass or good green hay. Gastrointestinal absorption of pyrimethamine may be delayed by simultaneous feeding (MacKay et al., 2000), so it is recommended that hay not be fed for 30-120 minutes after drug administration. In many management situations this delay is impractical, and there are no data to prove that simultaneous feeding and drug administration decrease the chances of successful treatment. To further complicate matters, if folic acid is to be supplemented, it should be given separately, several hours apart from drug administration intervals, to prevent drug interference with gastrointestinal absorption of the supplemented folic acid.

Treatment rarely has adverse side-effects. Some horses develop soft stools, probably due to the antibacterial effects of the sulfonamide, but this diarrhea is mild and self-limiting. Very rarely, foals born to mares treated during pregnancy may show signs of folic acid deficiency (Toribio et al., 1998). These signs include low red and white blood cell counts leading to weakness and inability to fight off infections, severe renal hypoplasia, and ultimately death in only a few days. Most of these foals were born to mares which received folic acid supplementation while undergoing EPM treatment. Mares' folic acid requirements may be 5-10 times higher in pregnancy than when not pregnant, but investigators have suggested that additional dietary folic acid given with pyrimethamine actually may inhibit absorption of most or all of the dietary folic acid,

resulting inadvertently in folic acid deficiency despite folic acid supplementation (Toribio et al., 1998). If there is concern over possible toxicity, weekly blood counts and/or folic acid determinations can be made on blood samples, but these tests cumulatively can become expensive. Horses already eating good quality fresh green grass or green hay are thought to consume sufficient dietary folic acid to prevent folic acid deficiency during treatment.

Recent epidemiological research has shown that "the likelihood of clinical improvement after diagnosis of EPM was lower in horses used for breeding and pleasure activities. Treatment for EPM increased the probability that a horse would have clinical improvement. The likelihood of survival among horses with EPM was lower among horses with more severe clinical signs and higher among horses that improved after EPM was diagnosed" (Saville et al., 2000b). In other words, conventional treatment works, improvement is a good prognosticator, and severity of clinical signs correlates with likelihood of response to treatment.

Newer drugs have been tried over the years with success. Diclazuril, toltrazuril, and ponazuril have been shown to have efficacy similar to that of conventional treatments (MacKay et al., 2000). Currently there are only two products FDA-labeled specifically for use in the treatment of EPM in horses. They are a 28-day paste formulation of ponazuril (Marquis®) and a pelleted form of diclazuril (Protazil®) for daily top-dressing of grain.

In acute and severe presentations, other supportive treatments may be necessary. Intravenous fluids are important if the horse is not drinking or eating. Analgesics (e.g., phenylbutazone) are given if trauma is suspected. In acute cases with brain signs, rabies cannot be ruled out easily so gloves are mandatory when handling the horse while awaiting the results of EPM testing. Corticosteroids (prednisolone, dexamethasone) are contraindicated since they may actually make the infection worse due to their ability to suppress the immune system.

Prognosis

Fewer than 1% of horses which are seropositive actually develop clinical signs of EPM. Early detection and treatment of EPM increases the chances of complete recovery (Saville et al., 2000a). Approximately 60-70% of treated horses return to their previous athletic function with no further abnormal signs. Approximately 10% of treated horses relapse after treatment is discontinued. At the first sign of a relapse, the horse should be re-examined and treatment should be re-instituted. Rarely horses will continue to test positive on CSF. In these horses, treatment may have to be continued for months or even years. Residual clinical signs after treatment may include varying degrees of ataxia, muscle atrophy, paresis, and focal cranial nerve abnormalities such as facial nerve paralysis or dysphagia (difficulty in swallowing).

Treatment of pregnant mares is sometimes necessary. If using conventional sulfa/ pyrimethamine therapy, the real risk of treatment is to the fetus, although some studies have shown this risk to be minimal especially early in pregnancy (Brendemuehl et al.,

1998). Supplementation with folic acid during pregnancy may actually increase risk to the fetus (due to impaired gastrointestinal absorption of folic acid after administration of both folic acid inhibitors and folic acid) (Toribio et al., 1998). Supplementation is not necessary if the mare is eating good quality grass or green hay routinely.

Prevention

The obvious method of prevention of EPM is to limit horses' exposure to opossums and their feces. Opossums should be kept out of the barn and especially away from sources of hay, feed, and water. It may even be necessary to keep cats or especially dogs loose in the barn to discourage "midnight raids" by opossums on the feed. Bagged feed may be safer than bulk feed, especially if the top of the bulk feed bin is open. However, the organism probably survives transport in bagged feed if the feed was contaminated before processing. Any shipment of feed or hay which seems to be contaminated with animal feces should be rejected. Extruded feeds seem to be most protective since the heat exposure during the extrusion process seems to kill the parasite before it is ingested by horses.

Preventative treatment of a normal horse which is in the same barn as another horse with a definitive EPM diagnosis is not recommended. It should be remembered that one infected horse cannot infect another normal horse; the parasite must come from the opossum, not from an infected horse. Unnecessary use of the medications to treat EPM may lead eventually to development of parasite drug resistance, making it more difficult to treat all EPM cases.

REFERENCES: Available upon request.

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