Question

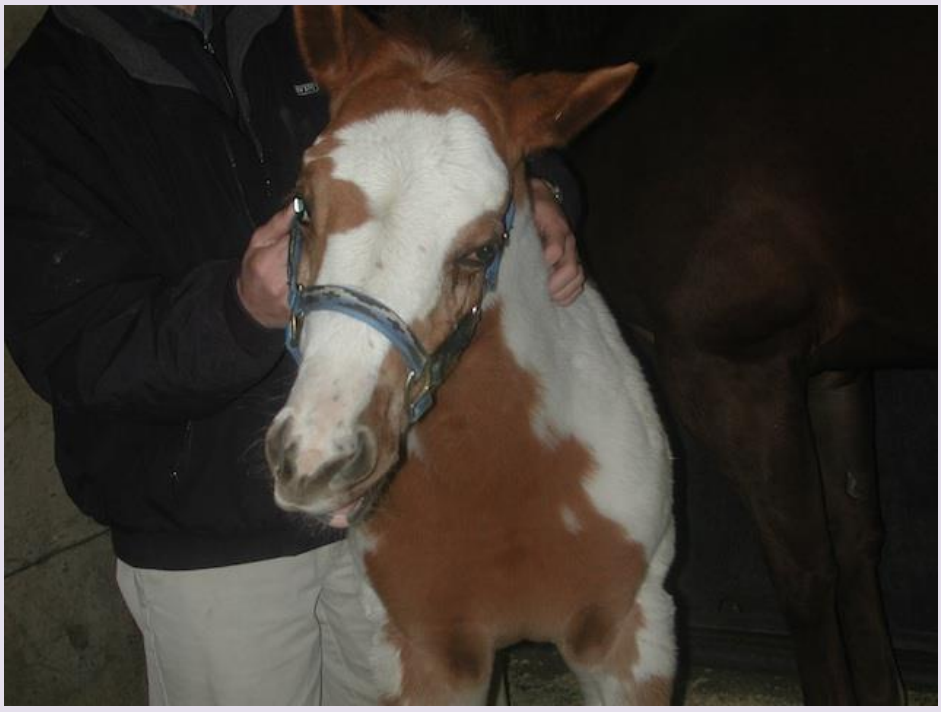
Which of these is the most appropriate treatment for a horse with equine protozoal myeloencephalitis?

- Ivermectin and fenbendazole
- Metronidazole and fenbendazole
- Penicillin and metronidazole
- Fenbendazole and praziquantel
- Trimethoprim-sulfa and pyrimethamine

Explanation - The correct answer is trimethoprim-sulfa and pyrimethamine. Equine protozoal myeloencephalitis is caused by Sarcocystis. This combination of drugs blocks folate metabolism by the protozoan at multiple steps and is generally agreed to be the most effective treatment for this condition. Recently, ponazuril has been approved and labeled for the treatment of EPM in horses and works by the same general mechanism.

Question

A 3-month-old paint colt presents to you after flipping over backward while being led by the owner the previous day. Clinical signs at the time of presentation are shown in the image (head tilt, flaccid ear, muzzle deviation). Based on the history and clinical signs, what cranial nerves are damaged and what is the most likely diagnosis?



- Cranial nerves V and VIII (left side); fracture of the sphenoid bone
- Cranial nerves V and VII (left side); atlanto-occipital malformation
- Cranial nerves V and VII (right side); atlanto-occipital malformation
- Cranial nerves VII and VIII (left side); fracture of the basisphenoid bone
- Cranial nerves VII and VIII (right side); fracture of the basisphenoid bone

Explanation - The correct answer is damage to cranial nerves VII (facial nerve) and VIII (vestibular nerve) on the left side caused by fracture of the basisphenoid bone. This is a common injury when a fractious young foal rears up and falls backward on the poll. The basisphenoid bone becomes injured resulting in injury to cranial nerves **VII** and **VIII**. Damage to the facial nerve results in the muzzle deviation (opposite direction of the side of injury, in this case deviated to the right), ptosis of the left eye and drooping of the left ear. Damage to the vestibular nerve results in the head tilt.

Question

Consumption of yellow star thistle results in this lesion.

- Nigropallidal encephalomalacia
- Destruction of the pons
- Destruction of the lateral and medial geniculate nucleus
- Leukoencephalomalacia of the reticular system

Explanation - The correct answer is nigropallidal encephalomalacia. Consumption of yellow star thistle destroys the globus pallidus and the substantia nigra. These lesions will result in a **characteristic dysphagia**.

Question

What structure is not likely to be affected with a guttural pouch infection of a horse?

- CN VII
- CN IX
- Carotid artery
- Cranial sympathetic trunk
- CN VI

Explanation - The correct answer is CN VI. This nerve does not run along the guttural pouch in contrast to the others listed. Both the internal and external carotids may be affected during a guttural pouch infection. In case you don't remember, the guttural pouch in a horse is the air-filled diverticula of the auditory tube which

communicates between the middle ear and the pharynx. Sometimes foreign material will get trapped there and result in infection.

Question

Hyperkalemic Periodic Paralysis (HYPP) in horses is caused by what autosomal dominant trait?

- Defect in voltage-dependent magnesium channels
- Defect in voltage-dependent sodium channels
- Defect in voltage-dependent potassium channels
- Defect in voltage-dependent calcium channels

Explanation - In horses with HYPP, the defect is in the sodium channel. In this disease, populations of sodium channels fail to inactivate and remain open. This, in turn, results in depolarization of the muscle membrane (closer to threshold) and hyperexcitability of the muscle. With further depolarization, the muscle cell membrane becomes unexcitable and paralysis may occur. The reason for the hyperkalemia is partially because of the movement of potassium out of the muscle cell as the myocyte repolarizes.

Question

Eastern equine encephalomyelitis is primarily transmitted by the _____.

- Fly
- Fecal-oral transmission
- Close contact with infected horse
- Tick
- Mosquito

Explanation - The correct answer is the mosquito. There have been over 27 different species of mosquitoes known to transmit EEE. After inoculation, it spreads via the lymphatics, and you don't usually see neurologic signs until approximately 5 days post infection. Both cortical and thalamic lesions result and they don't necessarily have to be symmetrical. Clinical signs may include head pressing, altered mentation, paresis, paralysis, convulsions, circling, ataxia, and death. Death may occur 2-3 days after the onset of clinical signs. These clinical signs apply to any of the forms of equine encephalomyelitis (VEE and WEE).

Question

A 7-year old Arabian gelding is presented to you for clinical signs of fever and lethargy of 2 days duration along with signs of hind-limb ataxia and muscle fasciculations of the face and neck. The CBC and biochemistry profile are relatively normal and you decide to collect cerebrospinal fluid (CSF) from the lumbosacral space. Results of CSF analysis yields the following:

Color Clear
Total Protein 156 mg/dL (reference interval 50-80 mg/dL)
Total Nucleated Cell Count 40 cells/mcL (ref interval less than 5 cells/mcL)
Cytology Lymphocytic pleocytosis

Based on the clinical signs and CSF analysis, which of the following is the most likely diagnosis and the most appropriate diagnostic test listed to confirm your diagnosis?

- Cervical Vertebral Malformation (CVM); Myelography of the cervical spine
- Verminous encephalitis (Micronema deletrix); Culture of CSF
- Equine Protozoal Myelitis (EPM); Western Blot of CSF
- Equine Herpes Virus (EHV) Myeloencephalitis; Virus isolation of buffy coat, nasal swab and/or CSF
- West Nile Encephalitis (WNV); Serum Immunoglobulin M (IgM) capture ELISA

Explanation - The correct answer is WNV. This is a mosquito born flavivirus that affects horses in multiple areas of the United States. Clinical signs are variable and can be mild (muscle fasiculations, slight ataxia) to severe (recumbency). Of note, muscle fasiculations is somewhat characteristic of WNV but fever may be detected in all patients. A readily available diagnostic test is the serum IgM capture ELISA which will detect infection, even in the face of vaccination. There is a vaccine available for WNV, making the clinical presentation less common.
With EPM you should not suspect fever.
With EHV1, you should suspect CSF analysis to be xanthochromic (effect of vasculitis), elevated protein with normal cell count.

Question

A 1.5 year-old Quarter Horse stallion presents to you for ataxia. The owner reports that he initially thought the horse may have been lame several weeks ago and the condition has progressed to his current state. You examine the horse and find muscle atrophy of the right quadriceps muscles as well as the left temporal-masseter muscles. The horse displays a spastic gait, worse on the right side. The gait seems to worsen when walking uphill. Cervical radiographs are within normal limits. Which of the following is an appropriate treatment recommendation for the most likely cause of the horse's clinical signs?

- Surgical vertebral stabilization
- Ivermectin and praziquantel
- Pyrantel pamoate
- Ponazuril

- Stall rest and phenylbutazone

Explanation - EPM can be tricky to diagnose because the clinical signs can be quite variable but the key findings are the "3 A's", namely **asymmetry**, **ataxia**, and **atrophy**. The onset of clinical signs are frequently gradual but rapid progression is seen in some cases. Differential diagnoses can include cervical vertebral malformation, equine degenerative myeloencephalopathy, equine herpes myeloencephalitis, polyneuritis equi, and verminous myeloencephalitis. However, the asymmetric and multifocal signs as well as the horse's age and gradual onset of signs make EPM the most likely diagnosis.

Treatment options for EPM include:

1) Ponazuril (Marquis) antiprotozoal paste
2) Diclazuril (Protazil) antiprotozoal pellets
3) Nitazoxanide (Navigator) antiprotozoal paste
4) Sulfadiazine-pyrimethamine combination

Surgical vertebral stabilization would be a treatment option for cervical vertebral malformation but this is less likely based on the asymmetric signs, temporal muscle atrophy, and normal cervical radiographs. Stall rest and phenylbutazone could be appropriate for certain orthopedic injuries. Ivermectin with praziquanel and pyrantel pamoate are dewormers but are not effective for EPM. Verminous myeloencephalitis is uncommon and typically has sudden onset with rapid deterioration and death.

Question

An 8 year old gelding Paint horse presents with a history of an inability to masticate. On physical exam the pterygoid, masseter, and temporalis muscles appear atrophied. Which cranial nerve was damaged?

- Facial
- Oculomotor
- Vestibulocochlear
- Abducens
- Trigeminal

Explanation - The correct answer is trigeminal. The trigeminal nerve is responsible for innervation to the muscles of mastication and sensation.

Question

A horse is suspected of having the muscle disease rhabdomyolysis, and you are seeking a laboratory test to help confirm the diagnosis. Of the following, which test would likely be the most helpful?

- Sorbitol dehydrogenase (SDH)

- Ionized phosphate
- Anion gap
- Gamma glutamyl transferase (GGT)
- Creatine kinase (CK)

Explanation - CK, also called, CPK, is found in muscle cells and is elevated in the serum when muscle damage occurs.

Question

A 1-year old Thoroughbred presents for a pre-purchase exam. On physical examination, you note a mild gait abnormality at the initiation of exercise which then diminished. Additionally, you noted that the horse was very heavily muscled. You decide to perform an electromyographic examination and obtain a crescendo-decrescendo signal of high-frequency repetitive bursts with a characteristic 'dive bomber' sound. What is your diagnosis?

- Exertional rhabdomyolysis
- Phosphorylase deficiency
- Hyperkalemic periodic paralysis
- Hypocalcemia
- Myotonia

Explanation - The correct answer is myotonia. These clinical signs and findings are consistent with a horse with myotonia. Etiology is not completely understood, but a genetic component is suspected. The **'**dive bomber**'** sound heard on EMG is produced by repetitive firing after contraction of affected muscle fibers and is essentially pathognomonic for myotonia. Hyperkalemic periodic paralysis would be seen in Quarter Horses, Paint horses, and Appaloosa horses rather than Thoroughbreds.

Question

A 3-year old Appaloosa mare presents with progressive neurologic signs over the past 2 weeks. She has an ataxic, spastic gait, knuckling on the left legs. She also has a right-sided head tilt and atrophy of the temporal and masseter muscles on the right side. There is muscle atrophy of the left gluteal muscles. Based on the most likely diagnosis, which of the following should you tell the owner about the prognosis for this horse with treatment?

- Most horses recover completely with treatment but a small subset of horses relapse when treatment is discontinued
- Most horses improve with treatment but relatively few make a complete recovery

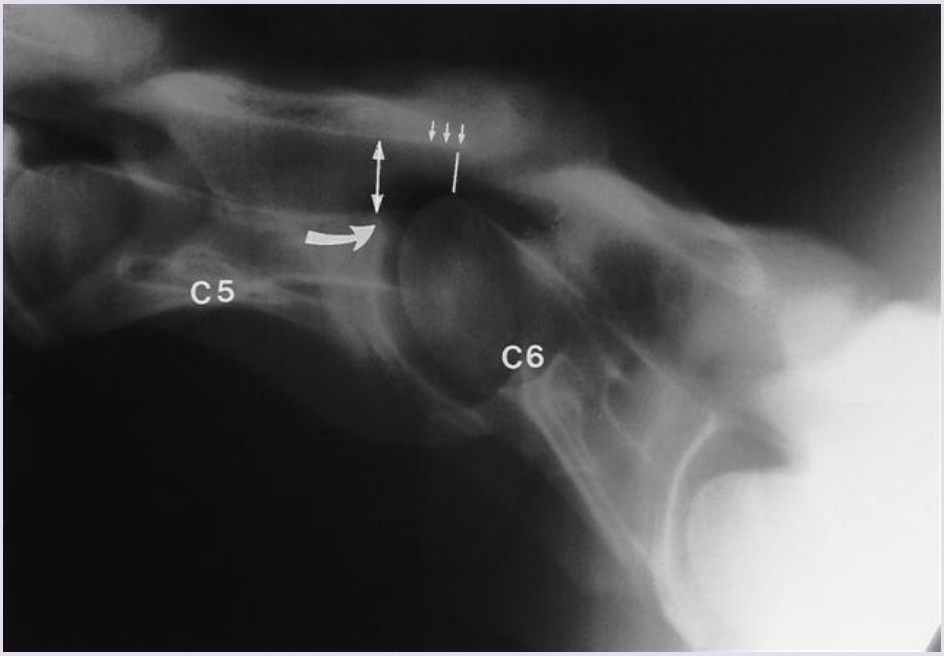
- Treatment will likely prevent further progression of the disease but the existing deficits are unlikely to improve
- It is unlikely that treatment will significantly delay the progression of this disease

Explanation - This is a case of Equine protozoal myeloencephalitis. EPM can be tricky to diagnose because the clinical signs can be quite variable but the key findings are the "3 A's", namely asymmetry, ataxia, and atrophy. The onset of clinical signs are frequently gradual but rapid progression is seen in some cases. Differential diagnoses can include cervical vertebral malformation, equine degenerative myeloencephalopathy, equine herpes myeloencephalitis, polyneuritis equi, and verminous myeloencephalitis. However, the asymmetric and multifocal signs as well as the horse's age and gradual onset of signs make EPM the most likely diagnosis.

The prognosis for horses with EPM is often described as variable. Studies show that about 70% of treated horses will improve but only up to 25% recover completely and perform normally. In addition, a subset (5-20%) of horses may improve while undergoing treatment but then relapse when treatment is discontinued. It is unclear whether relapse represents reemergence or persistence of resistant parasite or re-exposure. Based on these features of EPM, the best answer choice of those listed is that most horses improve with treatment but relatively few make a complete recovery.

Question

The radiograph illustrates an equine neurologic disorder. The neck is flexed to demonstrate a narrowing of the intervertebral canal at the junction of C5 and C6. What is this condition?



- Shaker foal syndrome
- Equine degenerative myeloencephalopathy
- Cervical vertebral stenotic myelopathy
- Canary grass staggers
- Ankylosing spondylitis

Explanation - Also known as Wobbler Syndrome or cervical vertebral instability, this condition results in symmetrical spinal ataxia.

Question

How long after receiving a puncture wound would you expect to see clinical signs of tetanus if a horse was infected?

- 10-14 days
- 1-2 hours
- 12-24 hours
- 3-4 weeks
- 3-5 days

Explanation - The correct answer is 10-14 days. Typically, the incubation period for tetanus is about 2 weeks, because it takes time for the spores to grow in an anaerobic environment before they produce toxin that can then be picked up by the nerves.

Question

A 9-year old Quarter Horse mare is presented for hindlimb ataxia, dog sitting (see image) and intermittent dribbling of urine. Two other horses at the same facility have recently demonstrated similar signs to varying degrees. What is the most likely cause?

- Cervical Vertebral Malformation (Wobbler Syndrome)
- Equine Protozoal Myelitis (EPM)
- Equine Herpes Myeloencephalopathy (EHM)
- Equine Degenerative Myelopathy (EDM)

Explanation - The most likely cause is EHM, which is associated with vasculitis of the central nervous system and associated clinical signs. Neurologic signs are usually acute and are characterized by hindlimb ataxia, hypotonia of the tail/anus, and urinary incontinence. EPM could possibly cause similar signs but is not typically seen in multiple animals on the same farm. Wobbler Syndrome and EDM typically affect young horses.

Question

How is equine herpesvirus 1 transmitted?

- Venereally
- Inhalation
- Fecal-oral

- Blood-sucking arthropods

Explanation - The correct answer is inhalation. Equine herpesvirus-1 or equine viral rhinopneumonitis is a rapidly-spreading disease that is spread by inhalation directly or indirectly from infected nasal discharge, aborted fetuses or placenta. EHV-3, the cause of equine coital exanthema is spread venereally. Both EHV-1 and EHV-4 are a cause of rhinopneumonitis, but they are very important because they also result in abortions. EHV-1 is also associated with myeloencephalitis and has resulted in various outbreaks in the U.S. and abroad. EHV-1 is the main cause of paresis, abortions, and neonatal foal deaths, according to a recent article by Patel and Heldens.

Question

A 4-week old foal is presented to you for evaluation of dysphagia and weakness. During your physical exam, you observe weakness and generalized muscle tremors. Upon further examination, you note weak tongue tone and dilated, non-responsive pupils. What is the most likely cause of these clinical signs?

- Guttural pouch mycosis
- Clostridium tetani
- Equine protozoal myeloencephalitis
- Clostridium botulinum

Explanation - The correct answer is Clostridium botulinum (also known as Shaker Foal Syndrome). Foals (2 weeks-6 months) are susceptible to the toxicoinfectious form of botulism, where they ingest the spores which grow in their intestines and make the toxin. The toxin blocks the release of acetylcholine from the neuromuscular junction, thus resulting in flaccid paresis or paralysis. Adults usually will only show clinical signs if they ingest the preformed toxin. Clinical signs are shaking, flaccid paralysis, drooling, decreased muscle tone, weakness, and dyspnea. The weak tongue tone is considered a cardinal sign. With medical treatment, prognosis is favorable; however prognosis is poor if treatment is not instituted, as many die of respiratory paralysis or pneumonia within days.

Question

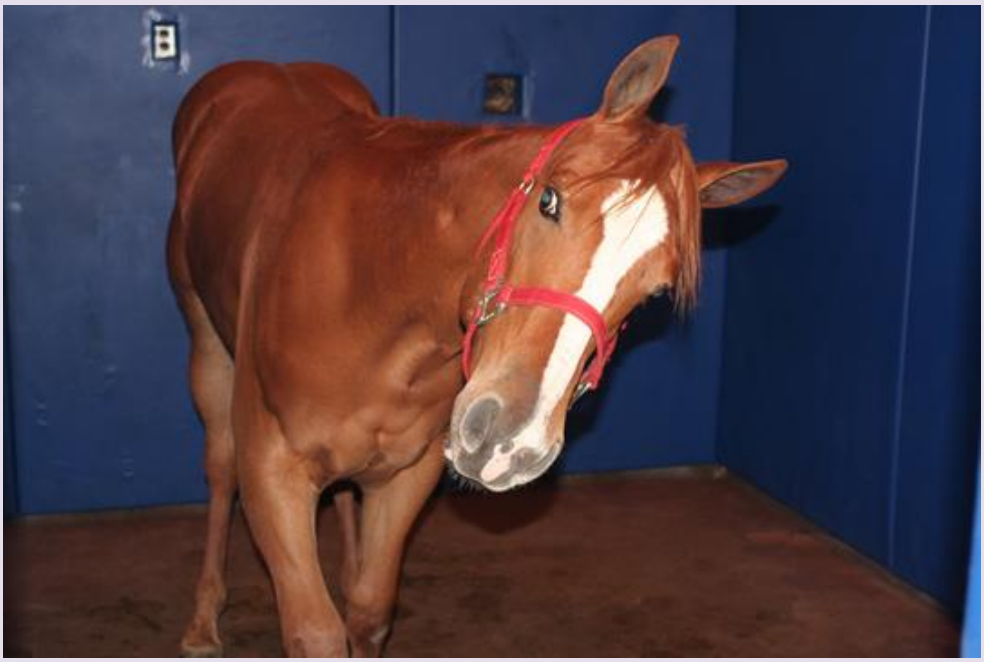
A horse presents for evaluation due to a progressive onset of what the owner describes as stiffness. On physical exam, the horse is indeed stiff. In fact, the horse is standing in a classic "saw horse" position. It is noted that the nostrils seem flared, and the tail is very stiff. The 3rd eyelid is prolapsed, and the horse seems particularly sensitive to sound and tactile stimuli. What is the etiology of this horse's condition?

- Close contact with infected horse
- Penetrating wound
- Ingestion of causative agent
- Inhalation of causative agent

Explanation - The correct answer is penetrating wound. To answer this question, you should realize that the clinical signs being described are classic for tetanus. Tetanus is caused by Clostridium tetani. Infection usually occurs via deep puncture wounds. Clostridium tetani releases a toxin which is capable of ascending up the nerves and into the spinal cord resulting in an ascending paralysis. The toxin blocks post synaptic inhibition to motor nerves, causing hypertonia and spasticity.

Question

You examine a 6 year old horse for head tilt to the left (left ear is lower, see image). The owner first observed it 2 days ago and it has since worsened. On PE, T=102F, and pulse and respiratory rate are normal. Other CNS signs include mild facial paralysis, mild ataxia, and nystagmus that does not change with head position. What is the most likely diagnosis?



- Locoweed poisoning
- Nigropallidal encephalomalacia
- Leukoencephalomalacia
- Vestibular disease
- Cerebellar abiotrophy

Explanation - These signs are compatible with mild infectious vestibular disease caused by bacterial otitis media/interna. Other possible causes of vestibular disease are guttural pouch empyema, polyneuritis equi, viral labyrinthitis, and traumatic skull fractures.

Question

An 18 year old mare presents with a head tilt, circling, and nystagmus with the fast phase to the left. Where is the lesion most likely located?

- Left Side
- Bilateral involvement
- Right side
- Not enough information

Explanation - The correct answer is right side. Although this is not right 100% of the time, the fast phase of nystagmus tends to go away from the lesion. The direction of nystagmus is named after the fast phase. So be careful, this can get a little confusing. In addition, if this question had provided information as to which direction the head tilt and circling were occurring, you would be that much more confident in localizing your lesion (ex. right-side head tilt or circling would be a right-sided lesion). Sometimes you may have paradoxical vestibular disease, in which the lesion does not follow the rules, and the lesion is on the opposite side of what is expected; however, this is rare. This is usually caused by destruction of the cerebellopontine angle or flocculonodular lobe.

Question

Which of the following is true regarding equine rabies vaccination in the United States?

- It is mandated by state law in all 50 states
- It is mandated by federal law
- It is required by state law only in endemic areas
- It is not required by law and only recommended in endemic areas

Explanation - Rabies vaccination in horses is only recommended in endemic areas and is not required by law. In endemic areas, boosters are usually performed annually.

Question

A client notices that her 8-year old Paint Horse gelding is lethargic, inappetant and looks to be in pain when forced to walk. Additionally, your client collected a urine sample for you to examine (see image). You collect a plasma sample to help determine the cause and it appears clear and normal. Which of the answer choices accurately describes a likely clinical scenario?

- Hemoglobinuria based on urine color and a normal-appearing plasma sample
- Extravascular hemolysis based on urine color and normal-appearing plasma
- Myoglobinuria based on urine color and a normal appearing plasma sample
- Normal urine based on the fact that horse urine has ample calcium carbonate crystals

Explanation - Myoglobinuria is characterized by brownish urine that does not clear on centrifugation along with normal-colored plasma. Myoglobin does not bind to serum proteins and is quickly excreted before reaching levels that would discolor the plasma. Also, the painful gait suggests a myopathy that may be related to the myoglobinuria. Conversely, hemoglobinuria is associated with a reddish discoloration of the plasma because hemoglobin is maintained in the plasma longer and is lost in the urine more slowly.

Question

An 11-month old Paint gelding is presented to you for symmetric ataxia, weakness, and spasticity of all limbs. When walking, the hind limbs frequently interfere with one another. Based on the signalement, history, and physcial examination findings, you suspect equine degenerative myeloencephalopathy (EDM). What diagnostic test would you use to support your suspicion?

- No definitive antemortem diagnostic test is available; low serum vitamin E levels are suggestive of EDM
- Collection and analysis of cerebrospinal fluid (CSF) demonstrating increased CSF protein and normal CSF nucleated cell count
- Biopsy of the sacrocaudalis dorsalis medialis muscle demonstrating muscle atrophy of type I muscle fibers
- Cervical radiographs and measurement of the saggital ratio

- Electromyogram (EMG) demonstrating diffuse increase in motor unit action potentials (MUAP) and positive sharp waves

Explanation - There is no definitive antemortem test for EDM; it can only be confirmed through histopathologic examination of the spinal cord and brainstem and the identification of diffuse neuronal fiber degeneration of the white matter. However, many cases of EDM have been associated with low serum vitamin E concentrations, so measurement of vitamin E is suggestive of disease. The exact etiology of EDM is unknown, however, oxidative stress and damage to the central nervous system is a prominent theory.

Question

You are called one day in August to examine a 7 year old mare that is kept pastured in a weedy field in California. She is thin and dull, has a normal TPR, and is neither pregnant nor lactating. In an effort to assess her appetite, you hold out some oats and she tries to eat them but cannot; her lips are pulled back and her tongue seems to tremor as she attempts to eat (see image). You believe this is some type of neurologic dystonia. What diagnosis should be strongly considered here?

- Brain abscess
- Rabies
- Protozoal myeloencephalitis
- Lead poisoning

- Yellow star thistle toxicity

Explanation - Centaurea solstitialis, aka yellow star thistle, grows in dry fields in California. Centaurea repens, or Russian knapweed, can also cause the same disorder. Some horses appear to avoid it; others appear to eat it with impunity, while some eat it and develop nigropallidal encephalomalacia. The loss of these upper motor ganglia results in loss of inhibition and dystonia, making it impossible for the animal to eat. Slow starvation results.

Question

A 3 month old Arabian foal presents for a progressive onset of intention head tremors, ataxia, dysmetria, and spasticity. On physical exam, the foal is noted to have proper mentation and is not weak. Heart rate, respiratory rate, and temperature are within normal limits. Given the signalment and presentation, what is the most likely diagnosis?

- Cerebellar abiotrophy
- Cerebellar dysplasia
- Sarcocystis neurona
- Inner ear disease

Explanation - The correct answer is cerebellar abiotrophy. Cerebellar abiotrophy is usually observed in foals which are less than one year of age, particularly 1-6 months of age. Cerebellar abiotrophy is the most common cerebellar disease found in horses. It is mostly seen in Arabian, Oldenburg, and Gotland breeds. There is no treatment, and signs may be progressive. Diagnosis is based on a good history and clinical signs such as intention tremors, lack of a menace, hypermetria, and ataxia.

Question

A 5 year old Standardbred from the east coast presents for progressive ataxia. On physical exam, there are areas of muscle loss noted around the left gluteal region of the horse. A neurologic exam revealed knuckling over on all four limbs, stumbling of limbs when walked in a circle, and a dysmetric gait. Given these clinical signs what is the most likely differential?

- Cauda equina neuritis
- Equine protozoal myeloencephalitis
- Cervical vertebral stenotic myelopathy
- Verminous myelitis

Explanation - The correct answer is equine protozoal myeloencephalopathy. If at any time you observe vague asymmetric/multifocal neurologic clinical signs with focal muscle atrophy, this disease should be your top differential. Other potential clinical signs include hyporeflexia, spasticity, localized areas of sweating,

cerebellar signs, head tilt, facial paralysis, circling, dysphagia, and blindness. EPM is caused by Sarcocystis neurona which is a protozoal organism that migrates randomly through the spinal cord and brain. This will result in damage of both white and gray matter.

Cauda equina neuritis is an inflammation of the nerve roots (usually the cauda equina but sometimes cranial nerves) which results in lower motor neuron signs. You may see paresis and paralysis. Clinical signs include chewing the tail head, hypotonic anus, fecal retention, urinary incontinence (see urine scalding of the thighs), and hindlimb ataxia. Unfortunately, there is no good treatment.

There are two forms of cervical vertebral stenotic myelopathy (aka Wobbler Syndrome). One form is called cervical vertebral instability. In this case, ventroflexion of the neck results in spinal cord compression. Most commonly affected sites are C3-4 and C4-5. The other form is known as cervical static stenosis. In this case, the compression is continuous regardless of the neck position. Clinical signs are usually in the form of a wide based stance, conscious proprioceptive deficits, ataxia, paresis, and spasticity which is worse in the hindlimbs.

Verminous myelitis results from aberrant migration of parasites through the central nervous system. Clinical signs are very similar to that of EPM. However, EPM occurrence is much more common. Parasites known to undergo aberrant migration in the horse are Strongylus vulgaris, Micronema deletrix, Draschia megastoma, and Setaria spp.

Question

A 2-year old Quarter Horse presents for intermittent muscle fasciculation followed by weakness. What condition should you suspect?

- Grass tetany
- Tetanus
- Myotonia
- Stringhalt
- Hyperkalemic periodic paralysis

Explanation - The correct answer is hyperkalemic periodic paralysis (HYPP). HYPP is seen in Quarter Horses due to a point mutation in a key part of a skeletal muscle sodium channel subunit. This results in elevation of the resting membrane potential to increase the likelihood of depolarizing. Excess concentrations of potassium can result in failure of the sodium channels to inactivate. Therefore, treatment is directed at decreasing dietary potassium.

Question

A 4 year old Quarter Horse presents to you for progressive neurologic signs of ataxia and paresis. You note asymmetric muscle atrophy of limb musculature. These findings are most consistent with which condition?

- Cauda equina neuritis
- Equine protozoal myeloencephalitis
- Wobbler syndrome
- Equine degenerative myeloencephalopathy

Explanation - The correct answer is equine protozoal myeloencephalitis. Equine protozoal myeloencephalitis (or myelitis) can affect any age horse and is caused by Sarcocystis neurona. It should be suspected in this case due to the asymmetry of clinical signs, as it is a multifocal disease of the central nervous system. Wobbler syndrome and degenerative myeloencephalopathy are diseases seen primarily in younger horses (< 3-4 years of age). Degenerative myelopathy causes a symmetrical ataxia. Cauda equina neuritis typically causes tail rubbing and urinary and fecal incontinence.

Question

You are examining a well-muscled 3-year old Quarter Horse stallion for muscle stiffness, difficulty walking, and prolapse of the third eyelid, followed by recumbency. The trainer informed you that the stallion has demonstrated similar clinical signs of lesser severity in the past. Based on this information, you suspect inherited disorder. What is the most appropriate therapy for this acute episode?

- Intravenous administration of 0.9% sodium chloride (NaCl) and 5% dextrose
- Intravenous administration of spironolactone (diuretic)
- Intravenous administration of 0.9% potassium chloride and 50% dextrose
- Intravenous administration of lactated ringer's solution (LRS) and enalapril

Explanation – The correct answer is IV administration of 0.9% sodium chloride (NaCl) and 5% dextrose. The hereditary disease you should suspect is hyperkalemic periodic paralysis. In acute episodes of HYPP, the clinician should try and drive down the serum potassium. This can be accomplished by administering potassium-free IV fluids (such as 0.9% NaCl) and medications (such as dextrose) to drive potassium into the cell. While LRS could be used when no other IV fluid is available, it does contain potassium.

Additionally, enalapril is an ACE inhibitor used for hypertension. One would clearly want to avoid IV solutions that contain high concentrations of potassium (KCl) or are extremely hypertonic (50% dextrose). Finally, while furosemide may sometimes be used to induce diuresis in some cases of HYPP, spironolactone is a potassium-sparring diuretic that would not decrease the serum potassium concentration.

Question

You are examining an ataxic horse and your differential diagnoses include EPM, cervical vertebral malformation, EDM, EHV1, polyneuritis equi, and verminous myeloencephalitis. Which of the following clinical presentations is most consistent with equine protozoal myeloencephalitis?

- Acute onset with concurrent fever and respiratory signs following abortions on a farm
- Lateralization of signs and concurrent neurogenic muscle atrophy of the quadriceps and gluteal regions
- Sudden onset and rapid progression of clinical signs
- Symmetric ataxia that is worse in the hindlimbs than the forelimbs
- Progression from hyperesthesia to anesthesia with progressive paralysis of the tail, rectum, bladder, and urethra leading to urine dribbling

Explanation - With EPM, the important signs to remember are asymmetry, ataxia, and atrophy. Lateralization of the signs (asymmetry) and quadriceps and/or gluteal muscle atrophy are most consistent with EPM.

Herpes myeloencephalitis is caused by EHV1 and often has an acute onset following an episode of fever, cough and nasal discharge or following abortions on a farm. This condition often affects more than one horse on a farm. The ataxia and weakness is usually symmetric.

Cervical vertebral stenotic myelopathy (CVM) and equine degenerative myeloencephalopathy typically cause symmetric signs with the hindlimbs usually a grade worse than the forelimbs. The signs of CVM may be worsened by flexing or hyperextending the neck.

Polyneuritis equi is more common in mature horses and usually starts with hyperesthesia progressing to anesthesia. There is progressive paralysis of the tail, rectum, bladder and urethra leading to urine dribbling.

Verminous myeloencephalitis is less common and the onset is usually sudden with rapid deterioration and death.

Question

How often should you administer tetanus antitoxin to a horse?

- Annually and 1 month prior to foaling
- After surgery or a wound in an unvaccinated horse
- At 3, 4, and 12 months of age, then annually
- To all foals and after any wound

Explanation - The correct answer is after surgery or a wound in an unvaccinated horse. The antitoxin should be used only when needed due to the risk of causing serum sickness or **Theiler's disease**. The toxoid should be given at 3, 4, and 12 months and then annually and 1-2 months prior to foaling. It should also be repeated after wounds or surgery in a vaccinated horse. The antitoxin should be reserved for horses that are unvaccinated and receive surgery or a wound. It should also be given to foals from an unvaccinated dam soon after birth. It can also be given in a horse showing signs of tetanus.

Question

A horse presents with vestibular signs. Which of the following findings allows the clinician to differentiate between central vestibular and peripheral vestibular disease?

- Head tilt
- Circling
- Proprioceptive deficits
- Nystagmus

Explanation - The correct answer is proprioceptive deficits. Observation of proprioceptive deficits helps localize the lesion centrally. In addition to proprioceptive deficits, any animal with multiple cranial nerve signs along with other vestibular signs is considered to have central vestibular disease. Nystagmus, circling, ataxia, and head tilt is seen in both central and peripheral vestibular disease.

Question

A 9 year old American Paint Horse presents to you for neurologic signs that have progressed over about 3 days. You have asked the owner about the horse's vaccination history and find out that the horse is vaccinated against rabies but not other pathogens. The horse was previously depressed and anorectic, progressing to head pressing and circling, and the horse is now recumbent. You perform cerebrospinal fluid analysis and find CSF protein of 80 mg/dl with a primarily mononuclear pleocytosis. Which of these is the most likely diagnosis?

- Equine protozoal myeloencephalitis
- Equine degenerative myelopathy
- Cervical vertebral malformation
- Eastern equine encephalitis

Explanation - The keys to this question are that the horse is unvaccinated for EEE, has a progression of central nervous system signs over several days and characteristic CSF changes.

Another reasonable differential would be rabies, particularly if the horse had not been vaccinated for it.

Equine protozoal myeloencephalitis (or myelitis) is usually seen in 1-6 year old horses and is caused by Sarcocystis neurona. It should be suspected when a horse has asymmetric neurological signs, as it is a multifocal disease of the central nervous system.

Cervical vertebral malformations and degenerative myeloencephalopathy are diseases seen primarily in horses under a year of age. Degenerative myelopathy causes a symmetrical ataxia.

Question

A horse with peripheral vestibular disease will not have this clinical sign.

- Facial nerve paralysis
- Left-sided head tilt
- Conscious proprioceptive deficits of the pelvic limbs
- Nystagmus

Explanation - The correct answer is conscious proprioceptive deficits of the pelvic limbs. Usually the CP deficits will be on the side of the lesion. So you can expect to see right-sided CP deficits on a right-sided central vestibular lesion. Head tilt, facial nerve paralysis, and nystagmus are non-specific signs of vestibular disease. However, they do help you determine on which side the lesion is located. Quick note: if there are multiple cranial nerves paralyzed then you should start thinking this could be central. A right sided head tilt, right sided nerve paralysis, and nystagmus with the fast phase to the left will indicate a right-sided lesion. Abnormal mentation or seizures along with other vestibular disease signs would imply central vestibular disease.

Question

A mare with an inability to blink and corneal ulceration indicates a lesion to this cranial nerve.

- Abducens
- Oculomotor
- Trochlear
- Facial
- Trigeminal

Explanation - The correct answer is facial (auriculopalpebral nerve). The facial nerve is responsible for providing motor innervation to the muscles of facial expression. Damage to this nerve may result in an inability to blink, muzzle deviation, ear droop, lack of nostril flare, and a loss of the menace and palpebral response.
The oculomotor nerve is involved in motor innervation to the dorsal, medial, and ventral rectus muscles. Additionally, this nerve innervates the palpebral levator, which is responsible for raising the upper eyelid.
The trochlear nerve innervates the dorsal oblique muscle of the eyeball. Injury to this nerve will yield a medial strabismus. The trigeminal nerve provides sensation to most of the face and also motor innervation to the muscle of mastication. The abducens nerve provides motor innervation to the lateral rectus and part of the retractor bulbi muscles.

Question

This neurologic disease has recently been associated with a deficiency in vitamin E.

- Dandy-Walker syndrome
- Cervical vertebral stenotic myelopathy
- Cerebellar abiotrophy
- Equine degenerative myeloencephalopathy

Explanation - The correct answer is equine degenerative myeloencephalopathy. The pathogenesis of cerebellar abiotrophy still remains unknown, although genetic, toxic, and infectious causes have all been incriminated. Cervical vertebral stenotic myelopathy is a developmental disease which is not associated with vitamin E deficiency. The disease results in either continuous or intermittent compression of the spinal cord at the cervical region. Equine degenerative myeloencephalopathy is the condition recently associated with vitamin E deficiency. Clinical signs are usually in the form of a wide-based stance, conscious proprioceptive deficits, ataxia, paresis, and spasticity, which is worse in the hindlimbs.

Question

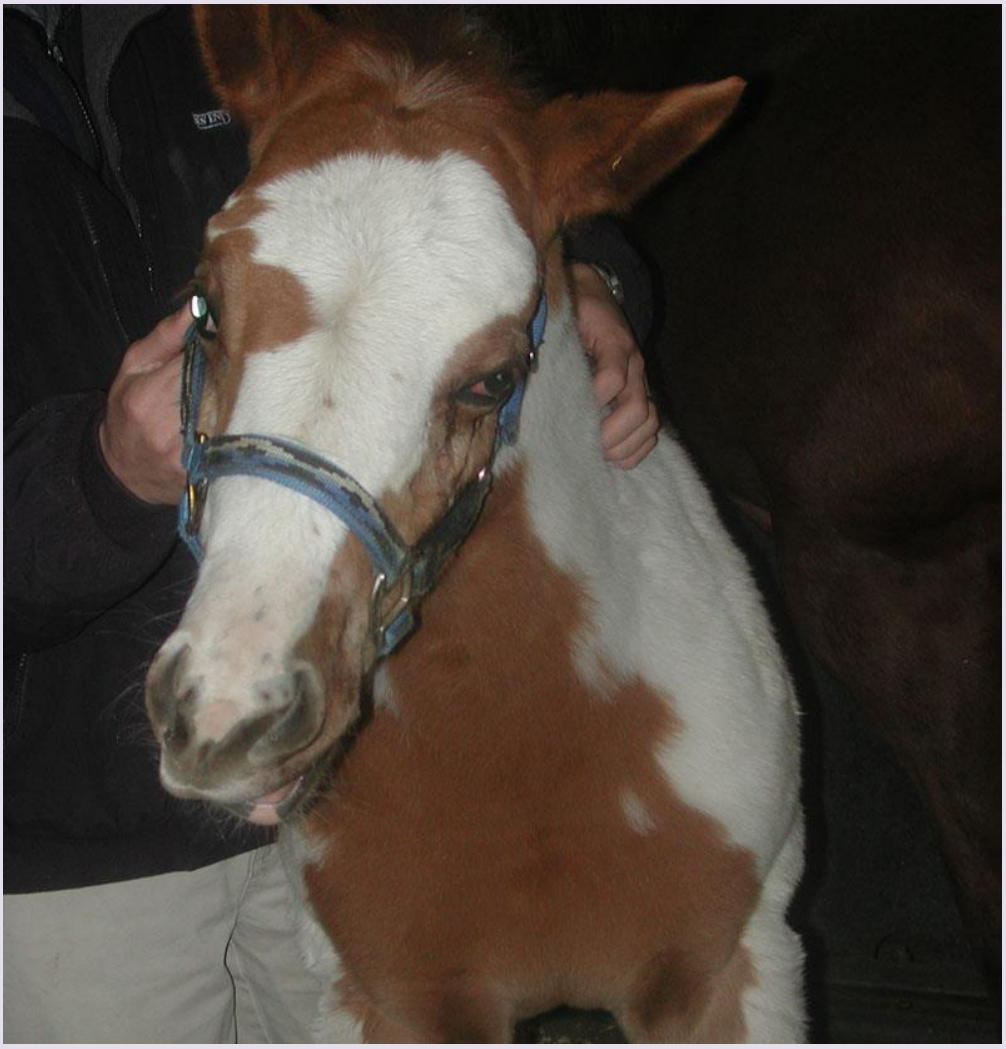
If a patient with vestibular disease exhibits a head tilt to the right, circling to the right, and conscious proprioceptive deficits on the left side, the lesion is on the _____.

- Left side
- Right side
- Not enough information
- Bilateral involvement

Explanation - The correct answer is left side. This patient is exhibiting paradoxical vestibular disease. Fortunately, there are proprioceptive deficits on the left side, and this allows you to localize the lesion the left. Paradoxical vestibular disease in horses may result with space-occupying lesions of the flocculonodular lobe or the cerebellopontine angle.

Question

You are called to examine a 7 month old colt that reared up and subsequently flipped over backwards and hit his head on the cement drive this morning while being put out to pasture. Based on the neurologic deficits observed in the image, you suspect trauma to what structure?



- Fracture to the occipital bone
- Fracture to the frontal bone
- Fracture to the hyoid bone
- Fracture to the basilar bones (basisphenoid/basioccipital)

Explanation - The image demonstrates a left head tilt, dropping of the left ear and deviation of the muzzle to the right. These clinical signs are suggestive of cranial nerve VII and VIII injury, which are located in close proximity to the basilar bones. The most likely fracture resulting in these lesions is fracture of the basilar bones, a common form of traumatic nerve injury in the foal.

Question

Cervical vertebral stenotic myelopathy in horses is best treated with _____.

- Non-steroidal anti-inflammatories

- Stall rest
- Surgery
- Steroids

Explanation - The correct answer is surgery. Surgery is required so that you may stabilize the cervical vertebrae and decompress the spinal cord. There are two forms of cervical vertebral stenotic myelopathy (aka wobbler syndrome). One form is called cervical vertebral instability. In this case, ventroflexion of the neck results in spinal cord compression. Most commonly affected sites are C3-4 and C4-5. The other form is known as cervical static stenosis. In this case, the compression is continuous regardless of the neck position. Clinical signs are usually in the form of a wide-based stance, conscious proprioceptive deficits, ataxia, paresis, and spasticity which is worse in the hindlimbs. Anti-inflammatories and stall rest may temporarily decrease clinical signs but are typically not a long-term therapy.

Question

A 4 month old male Arabian presents with a one-week history of ataxia, hypometria, conscious proprioceptive deficits, and generalized weakness. On physical exam, there is no muscle atrophy or cranial nerve deficits and a normal mentation. Additionally, the temperature is 100.2F. Which of the following is not a likely differential?

- Equine protozoal myeloencephalitis
- Equine degenerative myeloencephalopathy
- Cerebellar abiotrophy
- Cervical vertebral stenotic myelopathy

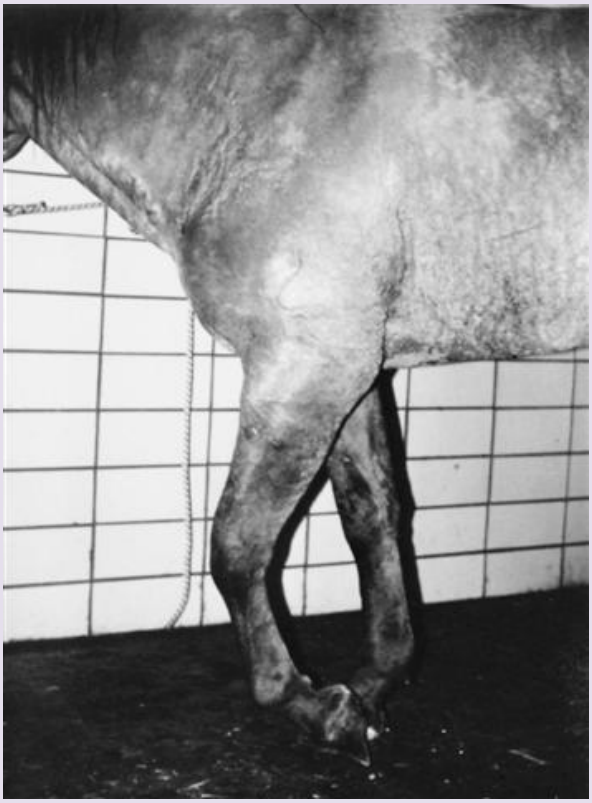
Explanation - The correct answer is cerebellar abiotrophy. The cerebellum is responsible for the coordination and regulation of range, rate, and strength of movement along with balance and posture. Clinical signs associated with cerebellar disease include intention tremors, hypermetria, hypometria, and ataxia. In addition, weakness is not observed with cerebellar abiotrophy. Mentation will be normal if the disease is strictly confined to the cerebellum. The key clinical sign not observed in this question is intention tremors.

The clinical signs and presentation are definitely compatible with equine degenerative myeloencephalopathy, cervical vertebral myelopathy, and equine protozoal myeloencephalitis.

Computerized tomography is needed to rule in or rule out cervical vertebral myelopathy. Histopathologic examination is the only way to definitively diagnose equine degenerative myeloencephalopathy (lesions in caudal brainstem nuclei and spinal cord). Serum and CSF immunoblotting is performed in order to determine exposure to equine protozoal myeloencephalitis, although EPM is unlikely in this young of an age.

Question

You are presented with a 5 year old gelding that stands with his left foreleg in the position shown, with dropped elbow and flexed carpus, fetlock, and digits. When he is walked, he drags the limb and is unable to advance the leg. The horse was placed in left lateral recumbency for a 4 hour surgical procedure to the guttural pouch yesterday. What is the correct diagnosis of his problem?



- Equine wobbler syndrome
- Ulnar nerve paralysis
- High radial nerve paralysis
- Brachial plexus tumor
- Equine dysautonomia

Explanation - The radial nerve is susceptible to damage from pressure when a large animal lays on it without adequate padding and circulation for a prolonged period. The advent of warm water beds for padding this area has greatly decreased the incidence. Rapid and aggressive treatment and excellent nursing care can allow some animals with radial paralysis to recover.

Question

A 1-year old Standardbred mare presents to you for evaluation of asymmetric ataxia and muscle atrophy of the gluteal muscles. Which of the following diagnostic test findings is most sensitive and specific for diagnosing this horse with equine protozoal myeloencephalitis?

- PCR of serum for Sarcocystis neurona DNA
- PCR of cerebrospinal fluid for Sarcocystis neurona DNA
- Immunoblot testing of serum for antibodies to Sarcocystis neurona
- Immunoblot testing of cerebrospinal fluid for antibodies to Sarcocystis neurona

Explanation - Immunoblot (Western blot) testing of cerebrospinal fluid in horses with neurologic signs is approximately 90% sensitive and specific. False positives on CSF can occur if the blood-brain-barrier is compromised and circulating antibodies from the serum enter the CSF. Serum immunoblot is indicative of exposure but not necessarily infection. PCR of CSF is only about 40% sensitive because the parasite is often in tissue and not free in the CSF or serum.

Question

A 9-year old racing Thoroughbred presents with clinical signs of lethargy, symmetric hind-limb ataxia, a hypotonic tail, urine dribbling, and a history of a fever. On physical examination, the horse's vital parameters are within normal limits but you do observe urine dribbling and ataxia. Upon rectal palpation the urinary bladder is enlarged and when pressure is placed on the bladder, urine is expressed from the urethra. The CBC and biochemistry profile are relatively normal and you decide to collect cerebrospinal fluid (CSF) from the lumbosacral space. Results of CSF analysis yields the following:

Color Xanthochromic
Total Protein 196 mg/dL (reference interval 50-80 mg/dL)
Total Nucleated Cell Count 6 cells/mcL (ref interval less than 5 cells mcL)
Cytology Occasional macrophages and lymphocytes observed

Based on the information provided, what is the most likely diagnosis and diagnostic test?

- Equine Protozoal Myelitis (EPM); Western Blot of CSF
- Verminous encephalitis (Micronema deletrix); Culture of CSF
- Cervical Vertebral Malformation (CVM); myelography of the cervical spine
- Equine Herpes Virus (EHV) Myeloencephalitis; Virus isolation of buffy coat, nasal swab, and/or CSF
- West Nile Encephalitis (WNV); Serum Immunoglobulin M (IgM) capture ELISA

Explanation - The correct answer is Equine Herpes Virus Myeloencephalitis. Clinical signs that are common with EHV myeloencephalitis include bladder paralysis with urine dribbling, fecal retention, and hindlimb ataxia. The xanthochromic CSF with the high protein and normal cell count (albuminocytologic dissociation) is also very suggestive of EHV myeloencephalitis. Virus isolation can be attempted on buffy coat samples, nasal swabs, and/or CSF in an attempt to identify the virus.

Question

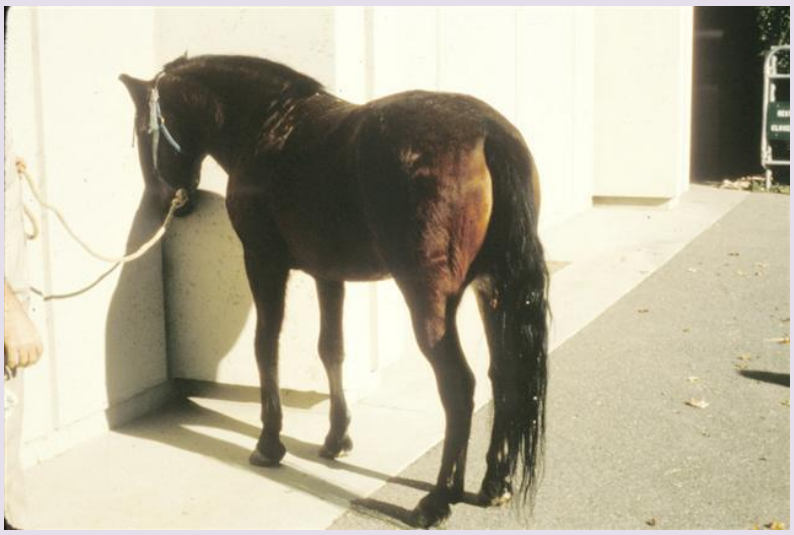
You are examining a 4-year-old racing Thoroughbred gelding for a 1-day history of reluctance to move. The gelding is bright and responsive with the only abnormalities noted on physical examination being tachycardia (56 beats/min), reluctance to move, and pain upon palpation of the muscles of the back and hindquarters (i.e. epaxial, gluteal, semimembranosus). The trainer states that this horse has shown these clinical signs a few times in the past. Based on initial examination, what initial clinicopathologic tests would be helpful in determining the most likely cause of the exam findings?

- Evaluation of creatine kinase (CK) and aspartate aminotransferase (AST) to rule-out myopathy
- Evaluation of blood urea nitrogen (BUN) and creatinine to rule-out renal disease
- Evaluation of sodium, chloride, potassium and calcium to rule-out a significant electrolyte derangement
- Evaluation of aspartate aminotransferase (AST) and gamma glutamyl transpeptidase (GGT) to rule-out liver disease

Explanation - Based on the clinical signs, the most likely cause is recurrent exertional rhabdomyolysis, commonly observed in racing Thoroughbreds. Typical clinical signs include muscle cramping of the major muscle groups; the cramping is painful and consequently causes the tachycardia, tachypnea, and profuse swelling. The muscle pain can be exacerbated upon physical examination by firm palpation of the larger muscle groups. The most sensitive serum enzyme to evaluate myositis is creatine kinase, a muscle specific enzyme with a short half-life. AST is not muscle specific but does become elevated at a slower pace, as compared to CK. Thus CK will rise and fall quickly whereas AST will increase and decrease slower once the myositis has resolved.

Question

A 4-year old horse has been losing weight over the last 4 weeks, is yawning frequently, and appears disinterested in and unaware of its surroundings, as shown in the photo. The horse has been vaccinated properly each year against West Nile, WEE/EEE, tetanus, flu, strangles, and EHV-1. The TPR is normal, but the horse can be positioned close to a wall and will remain there. You suspect what condition as most likely?



- Rabies
- Equine protozoal myeloencephalitis
- Western equine encephalomyelitis
- West Nile virus encephalomyelitis
- Hepatoencephalopathy

Explanation - Regardless of the cause of hepatic disease, when enough liver function is lost, hepatoencephalopathy can result. Increases in ammonia, aromatic amino acids, and mercaptans may play a role in developing CNS signs. The duration of 4 weeks and the normal TPR make an infectious disease less likely. The clinical signs do not fit rabies or protozoal myeloencephalitis.

Question

Which of the following medications is an FDA-approved treatment for equine protozoal myeloencephalitis (EPM)?

- Pyrimethamine
- Trimethoprim Sulfa
- Ponazuril
- Toltrazuril

Explanation - Ponazuril (trade name: Marquis) is an FDA-approved drug for the treatment of EPM. Ponazuril is an anticoccidial compound with cidal activity against Sarcocystis neurona. Historically, trimethoprim sulfa in COMBINATION with pyrimethamine has been used to treat EPM but is not specifically approved for this use.

You should probably be aware of both of these treatments as it can take the board several years sometimes to catch up to new treatments such as Ponazuril.

Question

What is the causative agent of equine protozoal myeloencephalitis?

- Neospora caninum
- Toxoplasma gondii
- Equine herpesvirus-1
- Sarcocystis neurona

Explanation - The correct answer is Sarcocystis neurona. Clinical signs of the disease vary in that they may be focal, multifocal, or diffuse in nature. One may observe focal muscle atrophy, and a neurologic exam may show ataxia and incoordination of all four limbs. However, you may only see one limb affected also. Muscle atrophy is most commonly seen in the quadriceps and gluteal regions of the hindlimbs. Horses may also have brainstem involvement and therefore exhibit a head tilt, facial paralysis, circling, and acute recumbency.

Question

An 8-year old Thoroughbred gelding was presented for acute onset of trembling, excessive recumbency, constant shifting of body weight while standing, and muscle atrophy. The horse is housed in a dry lot with no pasture availability. Diet consists of moderate quality grass hay and sweet feed. The CBC is normal with abnormalities on biochemistry analysis including mild elevation in creatine kinase (CK) and aspartate aminotransferase (AST). Cerebrospinal fluid (CSF) analysis shows no remarkable findings. Based on this information, what disease process do you suspect?

- Equine Herpes Myeloencephalopathy (EHV-1)
- Cervical Vertebral Malformation (Wobbler Syndrome)
- Equine Motor Neuron Disease (EMND)
- Equine Protozoal Myeloencephalitis (EPM, Sarcocystis neurona)
- Verminous encephalomyelitis (Halicephalobus deletrix)

Explanation - The correct answer is EMND. This is a neurologic disease associated with dietary vitamin E deficiency which is believed to contribute to oxidative damage to the CNS. However, the precise pathophysiology is unknown. Retrospective studies have noted that horses with EMND are commonly housed on dry lots with little to no availability to pasture or good quality hay (perhaps contributing to low blood vitamin E concentrations). The clinical signs in this question are classic for EMND whereas horses afflicted with the other options do not typically demonstrate trembling or shifting of body weight.

Question

Which of these would you least expect to see in an adult horse with tetanus?

- Quidding

- Extensor rigidity
- Profuse sweating
- Extended tail
- Tongue hanging out

Explanation - The answer is tongue hanging out. With tetanus, horses often develop lockjaw and although they may quid (drop food) or have dysphagia, their tongues do not hang out; this is seen in botulism. Signs of rigidity are due to the tetanus neurotoxin blocking post-synaptic inhibition to motor nerves. Other signs of tetanus include 3rd eyelid prolapse, pointed and erect ears, dyspnea, and muscle spasms.

Question

A 1.5-year old Quarter Horse gelding is presented to you for symmetric ataxia, weakness, and spasticity of all limbs, but worse in the hind limbs. When walking, the horse frequently drags his toes and the hind limbs frequently interfere with one another. Based on the signalment, history and physical examination findings, which of the following is the most likely cause of these clinical signs?

- Equine Protozoal Myeloencephalitis (EPM)
- Equine Motor Neuron Disease (EMND)
- Cauda Equina Syndrome
- Equine Degenerative Myeloencephalopathy (EDM)

Explanation - The horse in this question has clinical signs most consistent with EDM; cervical vertebral malformation (wobblers) is also a possibility, but was not provided as an answer. The cause of EDM is unknown, but this disease typically affects young horses (< 2-3 years of age; but older horses can develop disease). Clinical signs are a result of diffuse neuronal fiber degeneration of various portions of the central nervous system. This disease has been associated with low serum vitamin E concentrations, suggesting that oxidative damage may play a role in the development of disease.

EMND is typically associated with muscle tremors, shifting of weight while standing, muscle atrophy and recumbency. Cauda equina syndrome causes analgesia of the perineum. EPM can cause a range of clinical signs, but is typically with asymmetric neurologic deficits.

Question

An 11 year old Quarter Horse mare presents to you for progressive neurologic signs over 4 days. The horse has been vaccinated for rabies only. Signs began with anorexia and depression but moved on to hyperesthesia, propulsive walking and a head tilt. Cerebrospinal fluid analysis shows CSF protein of 75 mg/dl with a mononuclear pleocytosis. Which of these is the most likely diagnosis?

- Western Equine Encephalitis
- Equine degenerative myelopathy
- Wobbler Syndrome
- Equine protozoal myeloencephalitis
- Thiamine deficiency

Explanation - The keys to this question are that the horse is unvaccinated for WEE, has a progression of central nervous system signs over several days and characteristic CSF changes.

Another reasonable differential would be rabies, particularly if the horse had not been vaccinated for it.

Question

A horse presents for further evaluation as a result of a progressive onset of strange behavior, according to the owner. On physical exam, the horse is noted to have a jerky and awkward motion when trying to initiate movement. The horse is noted have an exaggerated arc and flight of the limbs when stepping over a curb. Where is the lesion most likely to be?

- Cerebrum
- Pons
- Midbrain
- Medulla
- Cerebellum

Explanation - The correct answer is cerebellum. The cerebellum is responsible for the coordination and regulation of range, rate, and strength of movement along with balance and posture. Clinical signs associated with cerebellar disease include intention tremors, hypermetria, hypometria, and ataxia. Mentation will be normal if the disease is strictly confined to the cerebellum.

Question

Which of these would you least expect to find in an adult horse that had ingested preformed Clostridium botulinum toxin?

- Mydriasis
- Dyspnea
- Proprioceptive deficits
- Tongue hanging out

Explanation - The correct answer is proprioceptive deficits. Proprioceptive deficits are not usually present with botulism because it is not a myelopathy but is a neuromuscular disease. The toxin blocks release of acetylcholine at neuromuscular junctions. The protruding tongue is a common early sign of botulism. Mydriasis occurs due to decreased pupillary response to light. Dyspnea occurs from paralysis of the diaphragm.

Question

Which of these would be appropriate for a horse with hyperkalemic periodic paralysis (HYPP)?

- Alfalfa hay
- Brome hay
- Beet molasses
- Timothy hay

Explanation - The correct answer is timothy hay. Of these choices, the only feed with low potassium is timothy hay. A low potassium diet is the most important nutritional modification in the treatment of HYPP. Regular exercise and feeding smaller, frequent meals can also reduce clinical signs. This disease is inherited in an autosomal dominant fashion, and owners should be discouraged from breeding affected animals.

Question

A race horse that has been overworked for the past week presents for a stiff gait, lethargy, anorexia, and oliguria. What is the most likely diagnosis?

- Pigment nephropathy
- NSAID toxicity
- Ischemic renal failure
- Pyelonephritis

Explanation - The correct answer is pigment nephropathy. Pigment nephropathy occurs as a result of myositis (tying up), which this presentation is consistent with. Nephrosis and subsequent renal failure is caused by large amounts of myoglobin being filtered by the kidney. This usually occurs in horses that have been subjected to extreme conditions such that the animal breaks down a substantial amount of muscle. The same condition can occur with intravascular hemolysis. In this case, the hemoglobin pigment is the culprit. However, this scenario seems to be less common in the horse.
