The neurological system

16

16.1 Disorders of the neurological system

Traumatic conditions

Spinal cord trauma

It is important that neurological trauma is identified and managed appropriately as equids with neurological damage can be a danger to themselves and other equids or people.

Clinical signs

- Ataxia – lack of coordinated muscle movements
- Postural deficits – wide-based stance, narrow-based stance, unstable
- Paralysis and muscle atrophy
- Weakness
- Increased or decreased muscle tone
- Hyperreflexia or hyporeflexia
- Loss of pain – superficial and deep pain. Loss of deep pain is a very poor prognostic indicator (this indicates extensive spinal cord injury).
- Loss of autonomic process – urination and defecation

The exact clinical signs depend on the location of the injury/lesion.

Treatment

Treatment is rarely recommended as the prognosis is poor for spinal cord damage. If the animal is recumbent, euthanasia is a necessity on welfare grounds. Recumbency will rapidly result in muscle damage from lying in one position. In cases of acute recumbency wait a minimum of 2 hours (with appropriate analgesia) before attempting to get the equid to stand. If severe neurological damage has occurred then the clinical signs will not improve in this time.

If, following an injury to the spinal cord, the animal is standing and showing gait abnormalities, euthanasia should again be considered on the grounds of welfare and safety.

If treatment is attempted, the equid should be able to stand, walk around without undue discomfort, and be able to pass urine and faeces. Rest the animal and administer steroids to reduce neurone swelling. Initiate a course of NSAIDs once the effects of the steroid have worn off to provide analgesia and long-term anti-inflammatory effects. If there is no improvement after 1–2 weeks consider euthanasia or retirement as the chances of a full recovery are much reduced. Recovery following nerve damage is often disappointing and slow.

Head trauma

Trauma to the head can result in neurological deficits if there is sufficient brain injury. Injuries to the poll commonly occur when a horse rears up and falls over backwards.

Clinical signs

- Injury to the head may be visible.
- Neurological signs due to brain injury
  - Altered consciousness, e.g. coma, dullness, depression
  - Convulsions, seizures
  - Ataxia
  - Head tilt, nystagmus, circling
  - Head pressing
  - Blindness

The clinical signs depend on the location and extent of brain injury.

Treatment

If there has been head trauma but the equid is neurologically normal, treat wounds symptomatically (clean, suture, analgesia and antibiotics as required). If there are marked neurological signs the safety of those handling the animal are the first priority. Sedate the affected equid if it is a danger to itself or others and, if the neurological effects are severe, consider euthanasia. Steroids can be used to reduce brain inflammation and swelling. Administer dexamethasone at 0.5 mg/kg IV or IM as soon after the original injury as possible.

Peripheral nerve trauma

Damage to peripheral nerves (lower motor neurons) results in very specific clinical signs in only the muscle(s) that the nerve innervates. If the nerve injury has resulted in bruising or swelling, rather than complete transection, a full recovery may be possible with anti-inflammatory therapy and rest. If the nerve has been fully transected full recovery is unlikely. Treat all cases of peripheral nerve damage with anti-inflammatory medication, steroids initially, followed by a course of NSAIDs.

Facial nerve paralysis

This is commonly seen when an equid lies with its head on the buckle of the head collar. Facial nerve paralysis can be a complication of general anaesthesia and it is always recommended to remove the head collar while the animal is recumbent.

Clinical signs

- Facial asymmetry – deviation of the nose to one side, dropping of the ear, lip, and eyelid on affected side
- Muscle atrophy (wasting), if nerve damage has been present for some time

Sweeny

This is seen in equids that pull carts or farm machinery with a yoke (a ring around the base of the neck). The yoke can cause pressure and injury to the suprascapular nerve as it crosses in front of the scapula. Ensure yokes are well fitted and padded.

Clinical signs

- Prominent scapular spine due to muscle atrophy
- Lameness and weakness in the affected limb

Obturator nerve paralysis

The obturator nerve, which passes along the internal aspect of the pelvis, may be damaged during a difficult foaling. If the equid is unable to stand, euthanasia should be considered on welfare grounds.

Clinical signs

- Hindlimbs do the 'splits' (splay out either side).

Infectious conditions

Tetanus

Tetanus is a global disease caused by Clostridium tetani, from the soil or faeces, entering the body via wounds. Once contracted it leads to a distressing and often fatal outcome (Kay and Knottenbelt 2007). Tetanus has been shown to be more prevalent in developing countries (Reichmann et al. 2008). Within the anaerobic environment of a deep wound the bacteria sporolate and produce the tetanus neurotoxin. The incubation period of the disease ranges from 3 days to 3 weeks. The neurotoxin potentiates normal sensory stimuli leading to muscular spasticity, hyperaesthesia, seizures and respiratory arrest.

Clinical signs

- Recent history of a wound – If no wound has been identified do not rule tetanus out if there is a strong indication from the clinical signs.
- Vague stiffness, mild signs of colic initially
- Muscle spasms – commonly the masseter muscles (known as 'lock jaw') and also muscles of the neck and hind limbs
- Spasms and stiffness which can be elicited by external stimuli such as loud noises and tactile stimulation
- Third eyelid protrusion, flared nostrils, erect ears and tail
- Fixed pupil dilation
- Extensor rigor 'saw-horse stance' with stiff rigid legs and hindlimbs stretched out behind (Figure 16.1.1)

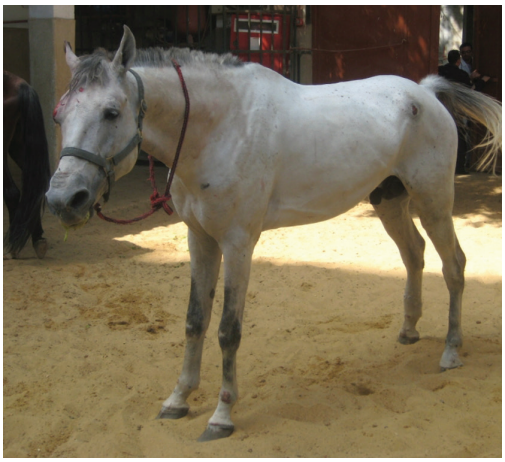

Figure 16.1.1 A horse showing classic signs of Tetanus: elevated tail, stiff-legged rigid stance.

- Collapse and death from respiratory arrest (usually within 3 to 10 days of the onset of clinical signs)

Diagnosis is based on wound history and clinical signs. The most commonly reported risk factor, in a series of affected horses, was the absence of vaccination (van Galen et al. 2006).

It has been suggested that donkeys have better survival rates than horses. Young animals are affected more often and more severely by tetanus than older animals. The combination of dysphagia, dyspnoea, and recumbency can be considered as an indicator of a poor prognosis in equids suffering from tetanus (van Galen et al. 2008).

Treatment

Treatment is sometimes successful in equids although, even with intensive therapy, mortality rates are reported at 68% and 75% in retrospective studies (van Galen et al. 2008, Green et al. 1994).

If treatment is attempted, keep the affected equid in a quiet, deep-bedded stable. Administer analgesia as muscle spasms are painful. Debride and lavage any wounds to allow oxygen into the space and reduce contamination. Administer high doses of systemic penicillin (2–3 times the normal dose rate for 7 days; however, be aware of the risks of antibiotic-induced colitis). The use of metronidazole is advocated in human treatment of tetanus (Gibson et al. 2009). Sedate the animal to induce muscle relaxation. Administer acetylpromazine, at a dose of up to 0.1 mg/kg body weight every 4–6 hours according to effect. Supportive therapy such as IV fluids and hand feeding will be necessary if the equid is unable to eat or drink.

If available, administer anti-tetanus toxin serum (immunoglobulin) as soon as possible after an injury, whether signs of tetanus are developing or not. These antibodies will bind the tetanus toxin before it can exert an effect on the neuromuscular junction. In human cases of tetanus, following the onset of clinical signs, intrathecal administration (spinal injection) of anti tetanus immunoglobulin is associated with higher survival rates (Kabura et al. 2006), although this has not been demonstrated in equids (Steinman et al. 2000).

If anti-toxin serum is not available, the vaccine may be administered after an injury. This will stimulate the immune system to generate anti-toxin antibodies; however, the response is unlikely to be fully protective until the booster 28 days after the initial vaccination. As the disease course for tetanus is usually less than 21 days this is unlikely to be an effective treatment in all cases.

Prevention

Tetanus toxoid is commonly administered to equids as a form of prophylaxis (Wilson et al. 2001). An initial course of two doses of vaccine at an interval of 4–6 weeks is required to achieve protection against tetanus for a 12-month period (Tasman and Huygen 1954). As the result of variation between individuals, a single dose may be effective in some cases but this cannot be relied upon to provide sufficient antibody levels for protection against the disease (Liefman et al. 1981). There is little evidence to demonstrate the duration of immunity; however, a booster every 1–2 years is recommended.

Botulism

Botulism is caused by the bacteria Clostridium botulinum which, in a similar way to tetanus, produces a neurotoxin. Unlike tetanus, the botulinum toxin causes flaccid paralysis. It is commonly associated with feeding silage/haylage which undergoes secondary fermentation when oxygen gains access to the forage. Rotting carcases, which may be eaten by equids browsing rubbish dumps, are another source of the bacterial toxin.

Clinical signs

- Progressive muscle weakness – which can be gradual or rapid in onset
- Difficult breathing – dyspnoea
- Tongue paralysis
- Eyelid drooping (ptosis) and pupil dilation
- Reduced anal tone, ileus, constipation, and urine retention
- Death due to respiratory paralysis or pneumonia after 24–72 hours

Treatment

Provide supportive therapy and administer the anti-toxin, if available. Identify and remove the source of the bacterial toxin to prevent botulism in other animals. Ensure only well-kept silage is fed to equids; discard silage that has been exposed to oxygen.

Rabies

Rabies is a fatal viral infection transmitted in saliva and predominantly spread by bites from infected wild or domestic animals including dogs and hyenas, and vampire bats in Central and South America. The rabies virus can also spread if infected saliva contacts mucous membranes or open wounds, and there is evidence of transmission through droplet inhalation.

The incubation period relates to dose and pathogenicity of the viral strain, immune status of host and proximity of bite to the central nervous system. The incubation period can be up to 1 year but signs of rabies usually develop within 3 weeks to 3 months of exposure. The virus can be present in saliva for up to 5 days before clinical signs appear.

Rabies is zoonotic and fatal to humans so extreme care must be taken when handling any suspected case (Figure 16.1.2)

All members of the veterinary team should have a course of pre-exposure rabies vaccinations. If you or any colleague suspects that they have been in contact with the saliva of a rabid animal, wash the affected area thoroughly with large amounts of soap and water and seek immediate advice from a medical doctor. Post-exposure vaccinations are recommended even if the individual had received a full course of rabies vaccinations prior to exposure.

Figure 16.1.2 Extreme care should be taken when handling any rabies case. Note the gloves, covering of the head to protect handlers from being bitten/contact with saliva, and the leg restraint.

Clinical signs

Cerebral or furious form (Figure 16.1.3)

Aggression, photophobia, straining, inappetance, chewing seizures

Brainstem or dumb form

Depression, ataxia, pyrexia, circling, head tilt, facial paralysis, dysphagia or anorexia with increased salivation, progressive ascending paralysis, flaccid tail/anus, urinary incontinence, self mutilation. Progressing to recumbency and a comatose state with death following within 5 days.

The dumb form is reported to be more common in equids than the cerebral/rabid form.

Always consider rabies in any rapidly progressing, multifocal neurological disorder.

Figure 16.1.3 The cerebral form of rabies.

Diagnosis

There is no ante-mortem test for rabies so diagnosis is based on history and clinical signs. A diagnosis can be confirmed through histopathological examination of central nervous tissue (brain tissue). Ensure compliance with local regulations for reporting rabies cases.

Treatment

There is no treatment for rabies. If the disease is strongly suspected, euthanasia should be carried out immediately with minimal handling of the animal (Figure 16.1.4). Handlers should wear protective clothing and gloves and use sufficient restraint to ensure that they are not bitten.

Figure 16.1.4 Carrying out euthanasia in a rabies case.

If the diagnosis is uncertain, the animal should be held in a secure place, with minimal handling, for observation. Any symptomatic treatment given to the animal must not compromise the safety of handlers. Visit the affected animal at its home to avoid risk to large numbers of people and animals at a clinic. It is absolutely imperative that owners are warned of the risks to themselves and their animals so that isolation procedures are strictly adhered to.

What do you do if an equid is bitten by a rabid animal?

If there is a history of previous vaccination, clean the wounds with antiseptic, revaccinate, isolate and watch for 45 days. Unvaccinated animals should be observed for 6 months in isolation, administer a course of rabies vaccinations (day 0, day 21 and day 56 following exposure).

Euthanase immediately if the bitten animal starts showing signs of rabies.

Rabies prophylaxis

Commercially available vaccines are available in many countries, and there may be government vaccination programmes in operation for the canine or wild populations. Equids should be vaccinated at 3 months old, 1 year old, then annually. Vaccinate mares before breeding rather than during pregnancy. There are a number of killed vaccines available for use in equines. Modified live vaccines should not be used.

> Any equine rabies vaccination programme will have little effect if the surrounding canine population is not effectively controlled or vaccinated.

Togaviral encephalitis

Eastern Equine Encephalitis (EEE)
Western Equine Encephalitis (WEE)
Venezuelan Equine Encephalitis (VEE)

The togaviruses are infectious, mosquito-borne diseases of equids affecting the central nervous system. Togaviral encephalitis in equids is largely confined to the Americas. All three diseases

are found in Central and South America, and EEE and WEE are mostly confined to the eastern and western United States respectively. VEE can cause large outbreaks of disease over extensive geographical areas in both humans and horses. Spread of this virus into Central America has had disastrous consequences. The virus is transmitted by mosquitoes, from reservoir hosts to equids. Infection is most common during the season when vector populations are greatest. WEE and VEE may also be transmitted horse to horse through nasal secretions.

The viruses are zoonotic, although not directly contagious, and notifiable to OIE.

Clinical signs in humans include fever, headache, stupor and seizures, and can lead to death. Inform owners to minimise contact with mosquitoes, and ensure your approach to the case is consistent with existing government protocols. Human vaccination is recommended for vets in endemic areas.

Clinical signs

The severity of neurological disease varies according to the type of togavirus but will include a generalised illness and fever progressing to behavioural change, circling, blindness, seizures, coma and death. Mortality rates are very high for EEE and VEE infections (40–100%).

Diagnosis

Diagnose by virus isolation or antigen detection in clinical cases. In the case of VEE, ELISA can be used for diagnosis.

Treatment

Administer supportive treatment including NSAIDs, fluid therapy and seizure control (diazepam 0.05-0.5 mg/kg IV as required); death is common if neurological signs progress.

Prevention

Vaccination with a killed trivalent vaccine is recommended in endemic areas in late spring (protection lasts for 6 months), and in the face of an outbreak. Mares should be vaccinated 1 month before foaling. Horses with VEE can be persistently viraemic and are therefore a source of infection.

West Nile Virus

West Nile Virus (WNV) is a mosquito-borne flavivirus; equids and humans are infected as dead-end hosts as the virus generally circulates in birds. WNV is normally distributed through Central and North Africa, the Middle East, west and central Asia and Australia. However, since 1999, the virus has spread rapidly across North America.

Signs of infection in equids vary from a mild fever to severe neurological signs progressing from mild ataxia to muscle fasciculation and cranial nerve deficits. Signs of cortical brain damage are rare.

Equine Protozoal Myeloencephalitis

This neurological condition is caused by the protozoa Sarcocystis neurona. Equine Protozoal Myeloencephalitis (EPM) results in a wide range of neurological signs depending on the migration pattern through the central nervous system.

Clinical signs

- Ataxia – can be severe
- Paresis, weakness and or spasticity
- Muscle atrophy
- Localised sweating
- Asymmetric pattern – The parasite migrates to a number of different locations in the nervous system.
- If untreated, it can progress to recumbency in 14 days to 6 months.

Treatment

The use of anti-coccidial agents, such as ponazuril, has been recommended (Mackay et al. 2008). NSAIDs can be given but corticosteroids are likely to worsen clinical signs. Consider euthanasia if signs deteriorate.

Case study – Tetanus in a working horse 16.2

Location Uttar Pradesh, India

Attending veterinarians Dr Hridesh Yadav and Dr Nidhish Bhardwaj

History

A 10-year-old mare, used for working in brick kilns, was having difficulty moving and feeding for 24 hours. The mare had been attended by a Local Health Provider on the previous day. The mare had no history of Tetanus Toxoid vaccination.

Clinical findings

Pulse Rate 48/min; respiration rate 24/min; CRT 2 Seconds; rectal temperature 38.4˚C; mucous membrane: pink; stiffness in the body and frightened attitude; erect ears and raised tail (figures 16.2.1 and 16.2.2); third eyelid 'prolapse' (figure 16.2.3)

Figure 16.2.1 Erect ears.

Differential diagnosis

- Tetanus
- Equine exhertional rhabdomyolysis
- Laminitis
- Hypocalcaemia
- Rabies
- Meningitis

On the basis of history and symptoms, the case was diagnosed as Tetanus.

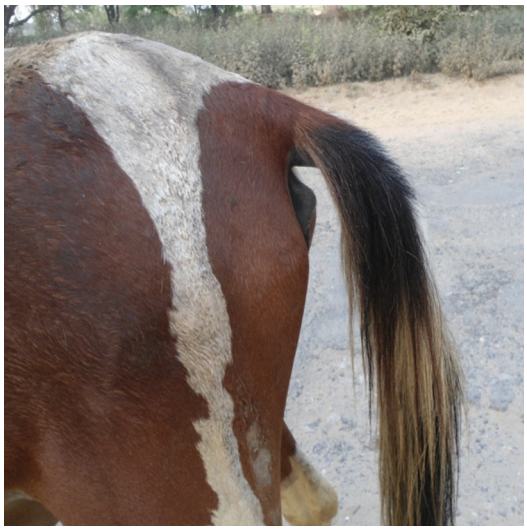

Figure 16.2.2 Raised tail.

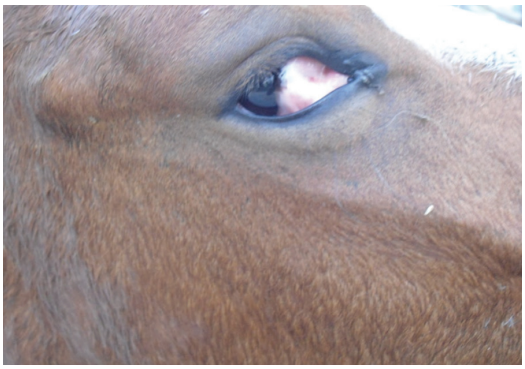

Figure 16.2.3 'Prolapsed' third eyelid.

Figure 16.2.4 The recovered horse at day 15.

Treatment

Treatment focused on three major aspects in tetanus: killing of organisms, neutralization of toxins and relaxation of muscles to prevent asphyxiation. Procaine penicillin (20,000 IU/kg, IM BID) was administered for 7 days. Penicillin is a drug of choice for Clostridial infections. Metronidazole (15 mg/kg B IV SID) was also administered for 7 days to kill anaerobic bacteria. Ketoprofen (2.2 mg/kg IV SID) was administered for 2 days and Acepromazine (0.10 mg/kg IV) on the first day, (0.08 mg/kg IM BID) for 5 days. Fluid therapy (Normal Saline and 5% Dextrose Normal Saline) was administered IV.

The owner was advised to take specific measures while nursing the animal at home including placing cotton plugs in the animal's ears to reduce sound-induced spasms, offering hand feeding with fresh green grass and bran, keeping the animal in a dark, quiet place with plenty of space, undertaking minimal handling of the animal to avoid muscle spasms and providing soft bedding to avoid self-inflicted injury.

The owner was also advised on prevention measures including: a full course of Tetanus Toxoid (TT) vaccine, as equines are very prone to Tetanus; vaccination of broodmares 4–6 weeks prior to foaling to ensure passive immunity for the foal; protection of working equines against wounds by proper saddle management and care; regular cleaning of stable.

Outcome

Fifteen days following initial examination, on re-examination the animal was fully recovered, moving and eating normally, see Figure 16.2.4.

Discussion

This case was successfully treated without the use of ATS (Anti Tetanus Serum) as the case was diagnosed in the initial stage and treatment started immediately. The case was treated with

antibiotics and supportive therapy. The toxins produced at the site are absorbed in the blood stream and attach to motor end plates. The toxins then pass to the CNS by crossing the blood brain barrier. Once the toxins are attached to the motor end plate they can never be released.

ATS intravenously, intramuscularly or subcutaneously does not cross the blood-brain barrier and has effect only on circulating toxin that has not attached to nervous system receptors. The amount of ATS applied intravenously does not have an influence on survival rates. These results are in accordance with other authors and corroborate their concern with the financial impact for poor animal owners by the use of high amounts of ATS without proven higher effectiveness (Reichmann et al. 2008).

ATS may be neither beneficial nor economically justifiable in the treatment of tetanus in working animals in the developing world. Early diagnosis, nursing care, high doses of parenteral penicillin and establishing aerobic conditions at the infected site are probably the most important aspects of treatment (Kay and Knottenbelt 2007).

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