Exotic diseases risk for UK horses

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Practice Notes

AS I wrote, the true extent of Schmallenberg virus was becoming evident, with the number of confirmed infected premises increasing on a weekly basis and the disease featuring on the national news and television's *Countryfile*.

Although relatively little is known about this virus, it seems to be carried by midges blown by wind to the UK from Europe. The cases of Schmallenberg infection seem to be mainly confined to south-eastern UK counties, but infection spread between incoming midges and the resident UK population could result in this disease becoming endemic.

The incident has highlighted the possibility that diseases traditionally believed to be exotic to the UK may start to be diagnosed here. Exotic diseases was a chapter I seemed to skip at veterinary school, so I've used this as an opportunity to refresh my memory.

Exotic infectious diseases can be classified into notifiable or non-notifiable. Notifiable diseases include West Nile virus (WNV), African horse sickness (AHS), equine viral arteritis (EVA) and equine infectious anaemia (EIA), while diseases including pirpoplasmosis and equine protozoal myeloencephalitis (EPM) are not.

Although procedures are in place to prevent EIA and EVA entering the country, occasional cases – in the form of carrier horses or uncertified semen – can potentially slip through the net. The hardest diseases to guard against are those borne by midges or mosquitoes. This will be especially important if the climate becomes milder in the UK. Midges are active at temperatures greater than 12°C and can be transported up to 700km by wind (Radostits, 2007a). In light of these factors, I have chosen to concentrate on some of the main midge and mosquito-borne diseases in the world.

WNV has caused intermittent outbreaks in recent years in north Africa, the United States and Europe, so there is potential for transfer to the UK equine population. Birds are the reservoir hosts, with disease transferred by *Culex* species mosquitoes (MacKay, 2009). Horses and humans are believed to be dead-end hosts, so infection spread relies on mosquitoes. Most outbreaks occur in summer and autumn in temperate climates, with a dramatic decline in cases after the first frost. However, the virus is able to overwinter in the midge population, so warmer winters may be influential.

WNV results in West Nile encephalitis (WNE), with clinical signs usually seen seven to 10 days after infection.

As with most causes of encephalitis, the symptoms are many and varied. About a quarter of horses presented will have mild to moderate pyrexia. The common neurological signs include ataxia and weakness, muzzle twitching, dullness or recumbency – although hyper-responsiveness, vestibular signs, dog-sitting, forelimb collapse, seizures or head pressing have also been reported (MacKay, 2009; Sloet van Oldruitenburgh-Oosterbaan, 2010). These cases can look very similar to EPM.

Definitive diagnosis of WNE is difficult. A presumptive diagnosis is often made if the horse exhibits associated clinical signs, especially muzzle twitching, and is within a confirmed area of infection. However, being one of the first UK vets faced with a suspected case would certainly not be an enviable task. Biochemical profiles will often show non-specific changes, including lymphopaenia, increased creatinine and muscle enzymes, together with electrolyte derangements. There is an ELISA available for identifying serum antibodies, but this identifies WNV infection and does not necessarily confirm clinical signs are due to WNE. A rising titre in paired serology samples can be useful to diagnosis cases in unvaccinated horses.

Interestingly, for every clinical case of WNE, about nine horses will show seroconversion with subclinical infection. The diagnosis would be supported by the cerebrospinal fluid (CSF) showing mild to moderate mononuclear pleocytosis and a low number of neutrophils. Identifying WNV-specific immunoglobulin M (IgM) in CSF will increase the likelihood of WNE. Definitive diagnosis can be achieved by isolating viral components from the central nervous system postmortem. Reports from the United States suggest 22 per cent to 44 per cent of cases will not survive (MacKay, 2009).

Treatment of WNE is largely symptomatic and providing supportive nursing care is vital. NSAIDs, dimethyl sulphoxide (DMSO) and vitamin E have all been used. Corticosteroid use remains controversial due to the potential of viral recrudescence after recovery (Radostits, 2007a). Although there is no scientific data available to support the use of interferon alpha or beta, anecdotal reports suggest it is a useful adjunctive therapy. Unfortunately, recovery can take months and the horse may never fully recover. In high-risk areas, disease control relies on vaccination and mosquito control.

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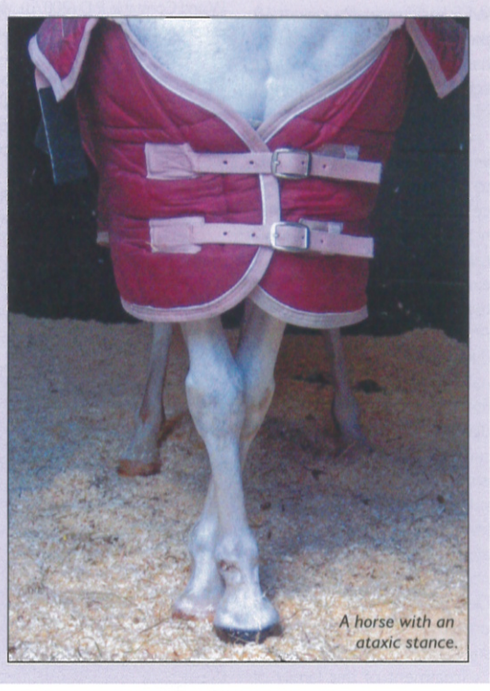

A horse with an ataxic stance.

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AHSV is endemic in southern and central Africa, yet outbreaks have been seen in more northern countries including Spain (Mair, 2002). This infection is spread via *Culicoides* midges and other arthropods, between host species, including equidae, elephants, camels, sheep, goats and also carnivorous species. It is not contagious, so direct horse-to-horse transfer does not occur. AHSV is an orbivirus; as is the bluetongue virus in ruminants. Infections outside the southern African continent are likely, due to movement of infected animals or wind movement of infected midges.

AHSV infection can result in various clinical presentations (Knottenbelt, 2010) depending on where the antigen localises. Clinical signs associated with the peracute or pulmonary form usually result three to five days after infection. They include marked pyrexia, dyspnoea, tachypnoea, severe coughing and frothy pink or yellow serous nasal discharge. Weakness and recumbency are likely to follow, with approximately 95 per cent of these cases dying within 24 to 96 hours (Radostits, 2007b). The subacute or cardiac form has a longer incubation period of seven to 21 days. Signs include oedema of the eyelids, supraorbital fossae, intermandibular area, neck, chest, lumbar and pelvic areas. Mild colic, restlessness, oesophageal paralysis and food regurgitation may be observed. If investigated, hydropericardium, endocarditis and pulmonary oedema may be detected. Only about 50 per cent of these cases will be fatal (Radostits, 2007b).

Acute or mixed African horse sickness will show clinical signs of cardiac and pulmonary forms. Mortality rate is between 50 per cent and 95 per cent of cases; most of which die within six days (Radostits, 2007b).

The mildest form is horse sickness fever, generally seen in donkeys and horses with partial immunity to AHSV. This form is usually confined to enzootic areas and cases will have moderate pyrexia, depression, inappetence and conjunctivitis. Most animals recover within eight days (Mair, 2002).

Diagnosis can be challenging. Blood samples may reveal leukopaenia, increased inflammatory proteins, muscle enzymes, creatinine and bilirubin. In cases that are rapidly fatal, serology may not confirm the disease. In horses that survive for 10 days, diagnosis may be confirmed using agar gel immunodiffusion, ELISA or other serological tests to detect antibodies. Histopathology and virus isolation in blood or tissues provides a definitive diagnosis. A PCR test is available that can help diagnose infection early in an outbreak. Postmortem histology of lung or heart tissue can provide a definitive diagnosis, as can viral PCR tests on lung, spleen or lymph node tissue.

Unfortunately, treatment again is supportive and symptomatic. A vaccine is available, but must be used in combination with reducing midge exposure. If infection occurs in non-enzootic areas, eradication can be achieved, as was achieved in Spain and Portugal in 1991(Radostits, 2007b).

EEV is another orbivirus transferred by *Culicoides* species, although it is less well documented (Radostits, 2007c). Equidae in southern Africa commonly have antibodies to EEV and, therefore, infection is probably widespread. It is suggested that many cases are subclinical; however, clinical signs may include pyrexia, lip oedema, neurological symptoms and enteritis. EEV has also been implicated in some cases of abortion. Diagnosis can be made using serum neutralisation assays or ELISAs, although definitive diagnosis is difficult. Other than symptomatic treatment, there are no specific therapeutic regimes or vaccines.

Eastern (EEE), western (WEE) and Venezualan (VEE) equine encephalomyelitis are neurotropic togaviruses usually diagnosed in the United States, Caribbean and central and southern America, often during the summer. This virus is similar to WNV in that it cycles between mosquitos and birds, and horses are generally considered to be dead-end hosts (Suarez-Mier, 2009).

The clinical symptoms of these togavirus infections are similar, but the mortality rates vary greatly. There is a variety of potential clinical outcomes and each horse may exhibit any or all forms of the disease. The mildest form is a low-grade viraemia and pyrexia. Other cases will show severe pyrexia, inappetence, depression, tachycardia and diarrhoea.

The final form of disease is clinical encephalomyelitis, which youngsters are most susceptible to. Signs include dullness, colic, head pressing, blindness, cranial nerve disease, ataxia, paresis and seizures (which will often rapidly progress to recumbency and death). EEE has a mortality rate of 75 per cent to 95 per cent, WEE has a mortality rate of 19 per cent to 50 per cent and VEE has a mortality rate of 40 per cent to 90 per cent (Suarez-Mier, 2009).

Again, diagnosis can be challenging. Haematology is likely to show some white blood cell derangements. Despite no characteristic biochemical changes, it is important to exclude hepatic encephalopathy from the differential list. An ELISA is available that can differentiate between vaccinal and virulent viral induced titres. Paired serology will help diagnose infection if the horse survives long enough. The CSF is not necessarily diagnostic, but is likely to show a high protein level and high numbers of nucleated cells; most of these will be non-degenerate neutrophils. Definitive diagnosis again relies on virus isolation from brain tissue (Suarez-Mier, 2009).

Treatment of this family of encephalomyelitis is purely supportive, using DMSO, NSAIDs, corticosteroids and interferon. Mortality rates are high and in the case of EEE, treatment is almost always ineffective. Vaccination and mosquito control is vital in areas where the disease is enzootic. If a horse recovers from clinical disease, it is likely to have immunity for at least two years (Radostits, 2007d).

Should any of these diseases be diagnosed in the UK, it will be imperative to consider reducing exposure to midge bites by using midge-proof stabling, including meshes over windows, preferably impregnated with insecticide. Stable location should be considered if building new housing – hilltop sites away from wet areas are obviously preferable. If possible, wet or marshy areas should be drained. Horses should be housed when particularly high numbers of midges are active (dawn and dusk). All this is useful to remember when considering the population of equines with sweet itch too.

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