Surra

Murrina, Mal de Caderas, Derrengadera, Trypanosomosis, El Debab, El Gafar, Tabourit

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Importance

Surra, caused by *Trypanosoma evansi*, is one of the most important diseases of animals in tropical and semitropical regions. While surra is particularly serious in equids and camels, infections and clinical cases have been reported in most domesticated mammals and some wild species. *T. evansi* is transmitted mechanically by various tabanids and other flies, and it can readily become endemic when introduced into a new area. The morbidity and mortality rates in a population with no immunity can be high. In the early 1900s, an outbreak in Mauritius killed almost all of the Equidae on the island. More recently, severe outbreaks have been reported in the Philippines, Indonesia and Vietnam. In addition to illness and deaths, surra causes economic losses from decreased productivity in working animals, reduced weight gain, decreased milk yield, reproductive losses and the cost of treatment.

Etiology

Surra is caused by the protozoal parasite *Trypanosoma evansi*. This organism belongs to the subgenus *Trypanozoon* and the Salivarian section of the genus *Trypanosoma*. Two genetic types of *T. evansi*, type A and type B, have been recognized. Most isolates worldwide belong to type A. Type B, which is not recognized by some diagnostic tests, has only been detected in parts of Africa as of 2015. Whether *T. evansi* should be considered a distinct species, separate from *T. brucei*, is controversial.

Species Affected

The principal hosts and reservoirs for *T. evansi* are reported to differ between regions; however, camels, equids, water buffalo and cattle are generally considered to be the major hosts among domesticated animals. Equids, Bactrian camels (*Camelus bactrianus*) and dromedaries (*Camelus dromedarius*) are highly susceptible to disease. Infections are usually mild or asymptomatic in cattle, water buffalo and related species in the Bovinae (the genera *Bos*, *Bubalus*, *Syncerus*, and *Poephagus*) in Africa or Latin America, but cattle and water buffalo regularly become ill in Asia. Many other mammals and marsupials are also susceptible to varying degrees; clinical cases have been reported in South American camelids, deer, sheep, goats, pigs, dogs, cats, tigers (*Panthera onca*), jaguars (*Panthera onca*), elephants, Sumatran rhinoceroses (*Dicerorhinus sumatrensis sumatrensis*), Himalayan black bears (*Selenarctos thibetanus*), coati (*Nasua nasua*), and experimentally infected wallabies (e.g., *Macropus agilis* and *Thylogale brunii*) and bandicoot rats (*Bandicota bengalensis*).

Infections have been documented in many wild mammals (e.g., various cervids, other large ungulates, wild pigs, lagomorphs, felids, canids, primates, small mammals) and marsupials, with or without disease, and some of these animals may help maintain *T. evansi*. In particular, vampire bats (*Desmodus rotundus*) are considered to be reservoirs as well as vectors in South America. Various small mammals, including capybara (*Hydrochaeris hydrochaeris*), have also been proposed as possible maintenance hosts. Some birds (e.g., young pigeons, chicks) can be infected experimentally, but their susceptibility in nature is uncertain.

Zoonotic potential

While *T. evansi* is not currently considered to be zoonotic, a few cases have been reported in humans. It is uncertain whether all of these infections occurred in people who are unusually susceptible or the disease is underdiagnosed.

Geographic Distribution

Surra is enzootic in Africa, the Middle East, many parts of Asia, and Central and South America. It also occurs in the Canary Islands of Spain, although control programs appear to have limited the organism to one small region.

Transmission

T. evansi does not require a biological vector. This organism, which can be found

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in blood and tissues, is transmitted mechanically by biting insects. Members of the deerfly and horsefly family, Tabanidae (e.g., the genera *Tabanus*, *Atylotus*, *Chrysops*, *Lyperosia*, and *Haematopota*) and flies in the genus *Stomoxys* are thought to be the most important vectors. Transmission by other biting insects (e.g., *Hippobosca* spp., mosquitoes in the family Culicidae, and midges in the family Ceratopogonidae) has been reported experimentally or suspected in the field, and might contribute to local spread. Sucking flies, such as *Musca* spp., may spread *T. evansi* when they visit contaminated wounds. Other organisms proposed as potential vectors include ticks and leeches, such as buffalo leeches in Asia.

Additional means of transmission include iatrogenic spread on contaminated needles or surgical instruments, and the ingestion of infected tissues by carnivores. Vampire bats can both maintain *T. evansi* and act as mechanical vectors in South and Central America. Transplacental transmission has been demonstrated in ruminants and donkeys, and transmission in milk and colostrum was reported in experimentally infected sheep. Trypanosomes cannot survive for long periods outside the host, and disappear relatively quickly from the carcass after death. In one recent study, organisms were detected as long as 13-15 hours in the heart blood of mice, although their viability had decreased to ≤ 5% by this time.

Disinfection

There is limited need for disinfectants, due to the fragility of trypanosomes in the environment, and no studies appear to have examined disinfectant susceptibility specifically for *T. evansi*. The closely related organism *T. brucei* can be inactivated by various agents including 0.05% sodium hypochlorite, 70% ethanol, 2% TriGene™, 0.1% hand soap, 2% formaldehyde and 0.05% glutaraldehyde. The temperature reported to kill 100% of trypomastigotes is 50°C.

Incubation Period

In the Equidae, the incubation period ranges from approximately one week to 2 months, with most cases appearing in 1-4 weeks.

Clinical Signs

Surra can be an acute, subacute or chronic disease, with the severity of the clinical signs differing between individual animals, as well as between species. Some animals die rapidly, especially among highly susceptible species such as horses and camels; in other cases, clinical signs may persist for months or years. Such chronic illnesses can occur in less susceptible species, but they may also be prevalent among equids and camels in endemic regions. Animals can also carry *T. evansi* subclinically.

Common clinical signs include fever (which can be intermittent in chronic cases), weight loss or wasting, lethargy, signs of anemia and enlargement of the lymph nodes. There may also be dependent edema, jaundice, petechial hemorrhages of the mucous membranes and abortions or stillbirths. Neurological signs have been documented in a number of species, especially in the late stages, and ataxia, with gradually progressive paresis of the hindquarters accompanied by muscle atrophy, is reported to be a common sign among horses in South America. Horses may have episodes of urticaria, and this sign was also reported in an outbreak among pigs. Testicular lesions in camels and experimentally infected goats suggest that, in some species, male fertility might also be impaired.

Additional signs documented in individual species include facial and laryngeal edema and ocular signs (e.g., conjunctivitis, keratitis, corneal opacity, anterior uveitis and/or hemorrhages) in dogs; diarrhea and conjunctivitis in some water buffalo; decreased milk production in dairy cattle; facial edema in Asian elephants; nasal hemorrhages in rhinoceroses; respiratory signs (dyspnea, coughing), diarrhea, ocular signs or arthritis in some experimentally infected goats; and diarrhea, vomiting, ocular signs, facial and limb edema, and external and internal abscesses in some experimentally infected cats. In addition, *T. evansi* causes leukopenia, which may result in immunosuppression and might decrease vaccine responses or exacerbate other conditions.

Post Mortem Lesions

The gross lesions tend to be nonspecific, and may include wasting or emaciation of the carcass, subcutaneous edema, signs of anemia, enlargement of the spleen, liver and lymph nodes, and petechiae on some internal organs. Muscle atrophy may be noted, particularly in the hindquarters. Icterus and nephritis may also be present. Fluid accumulation in other body cavities (e.g., ascites, hydrothorax) is sometimes seen. Cardiac lesions including hydropericardium, pericarditis and evidence of cardiomyopathy or myocarditis occur in some animals. The lungs may also be affected in some species; respiratory lesions (congestion, consolidation, edema, emphysema, hemorrhages and/or pneumonia) were reported in some *T. evansi*-infected cattle and water buffalo and some experimentally infected bandicoots, rats, mice and coatis.

In some horses with neurological signs, the cerebral hemispheres may be swollen and the gyri flattened. There may be severe edema and malacia, with the white matter becoming yellow, gelatinous and friable. Subpial hemorrhages may also be present.

Diagnostic Tests

A presumptive diagnosis may be possible if organisms consistent with *T. evansi* are detected in the blood, lymph nodes, tissues (e.g., at necropsy) or edema fluid by direct examination, and other trypanosomes do not exist in the area. While *T. evansi* differs in morphology from some other trypanosomes, it cannot be distinguished from certain species such as *T. equiperdum*.

In addition, atypical forms have been observed in some outbreaks (e.g., organisms that resembled *T. vivax* during the 2008 surra outbreak in Spain). Blood should be collected from live animals during a febrile period. The organism may be difficult to find, especially in mild or subclinical cases, and parasitemia is often intermittent in chronically infected animals. Repeated sampling may be necessary.

Microscopy specimens used to look for trypanosomes include wet blood films, used to detect the motile organisms, and thick or thin stained blood smears. Thick films have the advantage of being able to detect parasites in low numbers; however, the morphology of the parasite is difficult to determine. Detection can be improved with parasite concentration techniques including mini anion-exchange chromatography, hemolysis methods that use sodium dodecyl sulphate (SDS) to destroy the erythrocytes (i.e., wet blood film clarification or hemolysis centrifugation), hematocrit centrifugation (Woo method) or the dark-ground/phase-contrast buffy coat technique (Murray method). The latter two methods rely on the concentration of trypanosomes near the buffy coat after centrifugation.

Polymerase chain reaction (PCR) assays are used in some laboratories. They can identify the organism to the level of the subgenus *Trypanozoon*, but cannot distinguish it from *T. equiperdum*. Recombinant DNA probes may also be employed, but are not in routine use.

In some horses with neurological signs, immunohistochemical staining could detect parasites in the brain, even when they were not visible in hematoxylin and eosin stained sections. This technique has also been used to detect *T. evansi* in the brains of cattle, hog deer and buffalo. Other antigen detection tests have also been published; however, the most recent World Organization for Animal Health (OIE) Manual of Diagnostic Tests and Vaccines did not currently consider any method to be sufficiently developed for routine diagnosis using blood or serum.

Serological tests include ELISAs and card agglutination tests (CATT). The CATT detects IgM and is particularly useful early in the course of the disease. The trypanolysis test may be employed to confirm positive results, and immunofluorescent assays can be used with small numbers of samples. All serological tests may not have been validated or standardized for a host species, and cross-reactions can occur with other trypanosomes.

Some serological and molecular tests, including PCR, may not detect the type B *T. evansi* variants reported from parts of Africa. Assays that can recognize type B isolates and/or distinguish types A and B include mobile genetic element PCR (MGE-PCR) and loop-mediated isothermal amplification (LAMP).

Animal inoculation studies in rats or mice may be used, if necessary, to detect low levels of parasites, such as when importing an animal into a surra-free regions. They are not generally recommended unless the need is critical.

Treatment

Surra can be treated with antiparasitic (trypanocidal) drugs. The efficacy and toxicity of a particular drug may differ between species. Depending on the drug dose and other factors, treatment may be clinically curative without completely eliminating the parasite, and relapses are possible. Drug resistance also occurs. Cases with neurological signs are very difficult to treat, although some newer drugs may cross the blood-brain barrier to some extent.

Treatment may or may not be permitted in countries where *T. evansi* is not endemic.

Control

Disease reporting

A quick response is vital for containing outbreaks in surra-free regions. Veterinarians who encounter or suspect a *T. evansi* infection should follow their national and/or local guidelines for disease reporting. In the U.S., state or federal veterinary authorities should be informed immediately.

Prevention

T. evansi is excluded from uninfected areas by quarantines and testing. This organism has often become endemic after its introduction into a new area, due to the large number of potential hosts and its ability to be transmitted mechanically by numerous biting insects. Nevertheless, eradication was successful in a few cases when the outbreak was recognized early. In some instances, such outbreaks were controlled by quarantines, movement controls and the slaughter of infected animals. In 2008, an outbreak among camels and equids on an isolated farm in Spain was eradicated by treating the infected camels, with isolation and monitoring in a closed stable for 6 years. Two equids were euthanized 4 months after treatment; however, treatment appeared to be effective in the camels. An outbreak in France in 2006 was also eradicated by isolating and treating camels but euthanizing seropositive sheep in the area.

In endemic areas, it is difficult to control the biting flies that transmit *T. evansi*; however, some animals may be protected with insecticides/ repellents, traps, insect screens/netting in stables, and/or other controls. In one recent outbreak, most cases occurred among horses kept in open paddocks, while horses in nearby fly-proof stables were spared. Flies are most infective soon after feeding on an infected host (e.g., in the first half hour), and the highest probability of transmission is to nearby hosts. Because tabanids are persistent feeders and do not usually leave one animal to bite another more than 50 meters away, it is also considered advisable to separate highly susceptible animals, such as horses, from herds of cattle, water buffalo or other

animals that may be subclinical hosts. In South America, animals should also be protected from vampire bats.

Antiparasitic drugs are used routinely to protect susceptible animals in some endemic regions. Carnivores and omnivores should not be allowed to eat the carcasses of infected animals. No vaccines are available.

Morbidity and Mortality

The severity of the clinical signs can vary with the strain of *T. evansi* and with host factors including previous exposures, stress, concurrent infections and general health. Horses and camels are generally considered to be the most susceptible species, and often develop severe illness, with high case fatality rates, even in endemic regions. Donkeys and mules are reported to have less severe signs than horses. In camels, surra is most common soon after weaning, but can occur in all ages. Severe outbreaks are especially likely when *T. evansi* is introduced into disease–free areas or susceptible animals are moved into endemic regions. Morbidity rates of 50% or greater, with comparable mortality, can be seen in some herds. Chronically or subclinically infected animals may relapse with parasitemia under conditions of stress.

Infections are often milder in other species of mammals; nevertheless, severe cases and deaths have been documented in many species including water buffalo, cattle, other domesticated animals and captive wild animals. Why surra is much more likely to become clinical among cattle and water buffalo in Asia than Africa or Latin America is uncertain; however, mortality rates ranging from 10% to >90% have sometimes been reported among bovids in Asia, especially where *T. evansi* was newly introduced. Among carnivores, clinical cases have been reported more often in dogs than cats, and can be severe. Some studies suggest that infections might be relatively common in this species. Approximately 2% of dogs tested by microscopic examination had trypanosomes in one endemic region in India, and 29% of dogs were seropositive in a survey in Brazil. Deaths are especially likely in untreated stray dogs, but can also occur despite treatment.

Public Health

Humans possess innate resistance against many species of trypanosomes, including *T. evansi*, due to the trypanolytic activity of the serum protein apolipoprotein L-I. Nevertheless, there have been a small number of human infections with atypical trypanosomes, including a few infections caused by *T. evansi*. Whether illnesses caused by this organism are underestimated or occur very rarely and under unusual circumstances is currently unclear.

One *T. evansi* infection was reported in 1977, in a laboratory worker exposed to contaminated blood. The symptoms included insomnia, memory loss, tachycardia and an enlarged liver, spleen, and lymph nodes, and resolved after treatment with an antitrypanosomal drug. While the parasite was identified only by morphology (which is not definitive), the diagnosis appears likely in this case. A definitive diagnosis was established in two naturally-occurring cases. One occurred in 2005, in a 45-year-old Indian farmer who had a genetic defect in apolipoprotein L1. His symptoms included intermittent fever, chills, sweats and neurological signs. The diagnosis was established by PCR and antiparasitic treatment was successful. The other case occurred in Sri Lanka in 1999, in a patient who had a headache and intermittent fever. While this case was not published in the scientific literature, a recent review article reported that the identity of the parasite had been confirmed by PCR. Four additional suspected cases, diagnosed only by parasite morphology, were reported from India. They included one person who died 2 days after admission to a hospital. Another suspected case (2010) was a cattle farmer in Egypt who had recurrent episodes of fever. He was reported to have been hospitalized and "successfully treated."

In 2005, a study conducted in the village of the farmer with the apolipoprotein L1 defect did not detect trypanosomes in the blood of any other humans, although some people were seropositive. The serological test used has not been validated in humans. In a study from Egypt, published in 2013, there was no virological evidence of *T. evansi* infection in camel owners, using either PCR or microscopic examination of blood smears, although 10-46% of their camels had evidence of infection. A new organization, the Network on Atypical Human Infection by Animal Trypanosomes (NAHIAT) was established in 2011, and is coordinated by the Institute of Research for Development (IRD) and the Center for International Collaboration on Agricultural Research for Development (CIRAD). Its purpose is to coordinate information and research about various atypical trypanosomes, including *T. evansi*, in humans.

Internet Resources

Center for International Collaboration on Agricultural Research for Development (CIRAD), France
http://www.cirad.fr/en

Institute of Research for Development (Institut de Recherche pour le Développement) France
http://www.ird.lk/

The Merck Veterinary Manual
http://www.merckvetmanual.com/

World Organization for Animal Health (OIE)
http://www.oie.int

OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals
http://www.oie.int/international-standard-setting/terrestrial-manual/access-online/

OIE Terrestrial Animal Health Code
http://www.oie.int/international-standard-setting/terrestrial-code/access-online/

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