Eastern, Western and Venezuelan Equine Encephalomyelitis

Eastern Equine Encephalomyelitis (EEE), Eastern Equine Encephalitis, Eastern Encephalitis

Western Equine Encephalomyelitis (WEE), Western Equine Encephalitis

Venezuelan Equine Encephalomyelitis (VEE), Peste Loca, Venezuelan Equine Encephalitis, Venezuelan Encephalitis, Venezuelan Equine Fever

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Importance

Eastern equine encephalomyelitis (EEE), western equine encephalomyelitis (WEE) and Venezuelan equine encephalomyelitis (VEE) are mosquito-borne viral diseases, found in the Americas, that cycle among wild vertebrates but sometimes affect humans and equids, and occasionally other animals. The predominant form of the illness and its severity varies with the host and virus, but clinical cases of encephalitis can have a high case fatality rate, particularly when they are caused by EEEV.

Etiology

Eastern, western and Venezuelan equine encephalomyelitis, as they are traditionally called in the veterinary literature, are caused by the respectively named viruses in the genus *Alphavirus,* family Togaviridae. The human literature generally uses the term encephalitis for these diseases, rather than encephalomyelitis, and the corresponding viral names (e.g., eastern equine encephalitis virus) are now the officially accepted names for these viruses, though both 'encephalitis virus' and 'encephalomyelitis virus' are still used informally.

Eastern equine encephalitis virus complex

The eastern equine encephalitis virus (EEEV) complex, which was previously considered to be a single virus with four lineages, has been separated into two viral species, eastern encephalitis virus (formerly lineage I of EEEV) and Madariaga virus (formerly lineages II, III and IV). EEEV is more virulent than Madariaga virus in some experimentally infected mammals and birds, and this also appears to be the case in humans, though not necessarily in naturally infected horses.

Western equine encephalitis virus complex

The western equine encephalitis virus complex contains western equine encephalitis virus (WEEV) and several closely related alphaviruses including Sindbis virus, Whataroa virus, Fort Morgan virus (and variants Stone Lakes virus and Buggy Creek virus), aura virus, and highlands J virus. WEEV is the only virus in this complex of significant medical or veterinary importance in the Western Hemisphere, though Fort Morgan and highlands J virus can also be pathogenic for some species. Sindbis virus and Whataroa virus, which can cause a febrile human illness with polyarthritis in the Eastern Hemisphere, are not discussed in this factsheet.

Venezuelan equine encephalitis virus complex

The Venezuelan equine encephalitis complex contains a number of viruses, classified into 6 viral subtypes (I to VI), some of which contain multiple antigenic variants or serovars. Venezuelan equine encephalitis virus (VEEV) is comprised of subtype I variants I-AB, I-C, I-D and I-E. Other named viruses include Mosso das Pedras virus (variant I-F), Everglades virus (subtype II), Mucambo virus (III-A, III-C, III-D), Tonate virus (III-B), Pixuna virus (subtype IV), Cabassou virus (subtype V) and Rio Negro virus (subtype VI). Bijou Bridge virus is a strain of Tonate virus that was detected in wild birds in the U.S. Rocky Mountains in the 1970s.

The viruses of the VEE complex can be divided into 'enzootic' (or endemic) and 'epidemic' (or epizootic) viruses, based on their usual behavior and amplifying hosts. Epidemic VEE viruses, which all belong to VEEV variants I-AB and I-C, are thought to arise sporadically from VEEV variants I-D and I-E. Epidemic VEE viruses are amplified in equids, and can cause extensive epidemics affecting both equids and humans, but apparently become extinct once the epidemic ends. Viruses in the enzootic group, which contains all of the remaining viruses (including VEEV I-D and I-E), are maintained in cycles involving wild animals, are not amplified in equids, and occur in limited geographic areas. They can affect humans, but with rare exceptions, they do not causes any significant illnesses in horses.

Species Affected

Eastern equine encephalomyelitis

Birds are thought to be the principal reservoir hosts for EEEV, though rodents and other small mammals might also amplify this virus. The relative importance of different

birds is incompletely understood; however, this virus is usually associated with swamps and marshes, and some passerines, wading birds (e.g., herons, egrets) and members of other avian orders are known to be competent amplifying hosts. The primary reservoir hosts for Madariaga virus are still uncertain, though small mammals are thought to play a more prominent role. Other mammals, reptiles and amphibians can also be infected with EEEV, and some reptiles, such as snakes, have been proposed to play a role in virus overwintering. Domestic mammals, including horses, are not important amplifying hosts, though some infected horses were found to develop transient viremia sufficient to infect mosquitoes in the laboratory.

Clinical cases mainly occur in equids, but they have also been seen sporadically in other mammals including sheep, cattle, South American camelids, dogs, pigs, and captive or free-living wildlife such as wolves (*Canis lupus*), white-tailed deer (*Odocoileus virginianus*) and a harbor seal (*Phoca vitulina*). Birds in endemic areas mostly seem to be infected subclinically, but outbreaks or sporadic clinical cases have been reported in diverse species including chukar partridges (*Alectoris chukar*), ring-necked pheasants (*Phasianus colchicus*), turkeys, ratites, whooping cranes (*Grus americana*), captive African penguins (*Spheniscus demersus*), egrets, glossy ibises (*Plegadis falcinellus*), southern cassowaries (*Casuarius casuarius*), a mute swan (*Cygnus olor*), a bald eagle (*Haliaeetus leucocephalus*) and a flycatcher (*Empidonax* spp.).

Western equine encephalomyelitis

Wild birds, including passerines, are the usual reservoir hosts for WEEV but this virus may also cycle in blacktail jackrabbit (*Lepus califomicus*) populations, and snowshoe hares (*Lepus americanus*) are capable of amplifying it. WEEV has been isolated from some wild rodents, with or without clinical signs, and antibodies have been found in other mammals such as pigs, reindeer (*Rangifer tarandus*), bison (*Bison bison*), pronghorn (*Antilocapra americana*) and red foxes (*Vulpes vulpes*). One study reported that attempts to infect pigs with this virus were successful, but cattle were resistant even to high doses. WEEV has also been isolated from some reptiles (e.g., snakes, tortoises) and frogs, and reptiles have been proposed as possible overwintering hosts.

Clinical cases are seen most often in equids, but they have also been reported in western gray squirrels (*Sciurus griseus*), California ground squirrels (*Citellus beecheyi*) and some birds, including emus, turkeys, pheasants and chukar partridges. Some species of squirrels, ground squirrels, field voles (*Microtus pennsylvanicus*) and kangaroo rats (*Dipodomys* spp.) became ill after experimental inoculation.

Other WEE complex viruses

Highlands J virus mainly seems to infect wild birds, but it was isolated from the brain of one horse with encephalitis. This virus can cause clinical cases in some experimentally infected birds including turkeys, young chickens and young partridges. Fort Morgan virus occurs in cliff swallows (*Petrochelidon pyrrhonota*) and house sparrows (*Passer domesticus*), and can affect house sparrow nestlings. Aura virus is not known to cause disease.

Venezuelan equine encephalomyelitis

Wild rodents and other small mammals are the usual reservoir hosts for enzootic VEE viruses, though birds may be involved in a few cycles. These viruses do not usually cause any illnesses in animals; however, one I-E variant caused outbreaks of encephalitis among equids in Mexico in the 1990s. Horses are not good amplifying hosts for any enzootic VEE viruses, including this variant.

Epidemic VEE viruses mainly cause illnesses in equids, which are also the primary amplifying hosts. There have been rare reports of clinical cases in other mammals, including pigs, cattle, goats, sheep, dogs and rabbits. Cattle, pigs and dogs have occasionally been found to develop viremia sufficient to infect mosquitoes, but these species are not considered to be important in virus amplification.

Zoonotic potential

Humans can be affected by EEEV, Madariaga virus, WEEV, epidemic VEE viruses and most enzootic VEE viruses, but there are no reports of clinical cases from highlands J or Fort Morgan virus. People infected with epidemic strains of VEEV, but not EEEV or WEEV, can develop viremia sufficient to infect mosquitoes.

Geographic Distribution

EEEV is mostly found in eastern North America, where it is particularly common along the Gulf and Atlantic coasts and in some midwestern states around the Great Lakes. This virus has also been detected in a few states west of the Mississippi, and in parts of South America. Madariaga virus has been found in Central and South America, especially along the Gulf Coast, and the Caribbean. WEEV occurs in the western U.S. and Canada, and in parts of South and Central America as far south as Argentina. Fort Morgan virus (with its variants) is widespread in North America, while highlands J virus has been detected in the eastern U.S.

Enzootic VEE viruses can be found in parts of the U.S., Mexico, South and Central America, with each virus generally occurring in a limited area. VEEV (subtypes I-AB to I-E) is absent from the U.S. and Canada, though some variants can be found in Mexico. Epidemic VEE viruses (VEEV variants I-AB to I-C) tend to arise most often in the northern regions of South America, but epidemics can spread into other parts of South and Central America, and occasionally into North America.

Transmission

EEEV, WEEV, highlands J virus and the members of the VEEV complex are mainly spread by mosquitoes. *Culiseta melanura* and *Culex tarsalis* are important vectors in the sylvatic cycles of EEEV and WEEV, respectively, in North America, while members of the genus *Culex* are prominent vectors for Madariaga virus and enzootic VEE viruses. However, many other mosquitoes can also transmit these viruses, and some may be more important in infecting

domestic animals and humans. Epidemic VEE viruses can be transmitted efficiently by a number of mosquito genera.

Other arthropods may also be involved occasionally. The cimicid swallow bug (*Oeciacus vicarius*), an ectoparasite of swallows, is the main vector for Fort Morgan virus, and was also found to harbor Bijou Bridge virus, an enzootic VEE virus. Mites or ectoparasites of birds might play a minor role in transmitting some other viruses during close contact, and blackflies were proposed as mechanical vectors for epidemic VEEV strains during some outbreaks. Ticks can be infected by both enzootic and epidemic VEEV strains, though their role in nature, if any, is unclear.

Non vector-mediated routes may occasionally play a minor role. Oral inoculation of EEEV has been demonstrated in pheasants, and other birds may also be susceptible to this route. Emus can shed large amounts of this virus in rectal and oral secretions and regurgitated material, while the presence of large amounts of EEEV on the feathers of pheasants suggests the possibility of transmission by pecking, feather picking or preening. Transmission via cannibalism or predation also appears possible in birds. Horses can shed epidemic VEE viruses in body fluids, though there are currently no reports of direct transmission between horses, or from horses to humans; while human cases have been documented after exposure to aerosolized debris from the cages of infected laboratory rodents. Person-to-person spread has never been reported, though VEEV is sometimes present in human pharyngeal and nasal secretions. However, transplacental transmission of WEEV and VEEV has been seen in pregnant women, and one EEEV-infected organ donor infected three solid organ transplant recipients.

How some of these viruses survive the winter in cold climates is still uncertain. Proposed mechanisms for EEEV and WEEV include prolonged persistence in birds, vertical transmission in mosquitoes, overwintering in reptiles and/or periodic reintroduction from warmer climates by migrating birds. Environmental survival is likely brief, though VEEV is reported to survive for a short time in dried blood and exudates. One experiment found that inactivation of 90% of this virus on glass took approximately 98 hours at room temperature (20-25ºC/ 68-77 ºF) in the dark. Whether viruses in the environment could infect animals or humans this long is unclear, as the experimental conditions were artificially optimized to recover the virus from the surface. There is little information about the persistence of EEEV and WEEV in the environment, but EEEV has been isolated from feather quills for up to 6 days.

Disinfection

As enveloped viruses, alphaviruses are expected to be susceptible to many common disinfectants including sodium hypochlorite, 70% ethanol, 3-6% hydrogen peroxide, 2% peracetic acid, phenolic agents, glutaraldehyde and formaldehyde. Madariaga virus in cell culture medium was inactivated within 5 minutes by heat of 95ºC (203ºF). It was also susceptible to UV light.

Infections in Animals

Incubation Period

The initial nonspecific signs of illness in equids can appear within a few days, while encephalitis generally becomes evident in about 5-14 days.

Clinical Signs

Clinical cases in equids

EEEV, WEEV and epidemic VEE viruses can infect equids subclinically, cause a febrile illness without neurological signs, or result in encephalitis, though EEEV is more likely to cause severe signs and the course of the disease may be shorter. Most horses that become ill initially have only a fever and nonspecific signs (e.g., anorexia, depression), but some subsequently develop encephalitis with signs of altered mentation (e.g., obtundation), hypersensitivity to stimuli, tremors or other involuntary muscle movements, impaired vision, behavioral changes (e.g., aimless wandering, head pressing, circling), an inability to swallow, ataxia, paresis, paralysis and/or convulsions. Periods of excitement or intense pruritus have been reported, and laterally recumbent animals sometimes have a characteristic paddling motion. Some affected animals may also have colic, diarrhea, constipation or significant weight loss. Affected horses can die within a few days, particularly when infected with EEEV, and horses that recover from encephalitis may have residual neurological deficits.

Enzootic VEE viruses typically infect equids subclinically or cause only mild, nonspecific clinical signs; however, one enzootic I-E virus in Mexico caused severe and frequently fatal cases of encephalitis.

Clinical cases in other mammals

Cases of EEE described in mammals other than equids have usually been characterized by neurological signs, often accompanied by fever and other nonspecific signs of illness. Infrequently reported signs included respiratory crackles or dyspnea, diarrhea (puppies) or excessive salivation (deer). Inappetence was common; however, one febrile young sheep remained alert and maintained a good appetite until it was euthanized due to progressive paralysis. While many cases progressed rapidly to the terminal stage, and sudden deaths have been seen, a longer clinical course is also possible.

Deaths have also been reported in various mammals including rabbits, goats, dogs and sheep during some VEE epidemics; however, these cases seem to be unusual, as attempts to reproduce the illness by experimental inoculation were only successful in rabbits; other species developed few or no clinical signs. WEEV was isolated from the brains of dead and moribund squirrels and ground squirrels in one outbreak, and the illness could be reproduced experimentally in some squirrels, ground squirrels, kangaroo rats and voles. Most of these animals had only nonspecific signs of illness before death, but some developed ascending paralysis.

Nonhuman primates infected with EEEV, WEEV or VEEV are usually inoculated via aerosols as animal models for weaponized viruses. These animals often develop fatal encephalitis from EEEV or WEEV, though VEEV generally causes only a self-limited, nonspecific illness, sometimes accompanied by neurological signs such as tremors or ataxia. However, one study that examined parenterally inoculated cynomolgus macaques, to mimic naturally-acquired infections, found that animals infected with VEEV or WEEV remained asymptomatic, while macaques inoculated with EEEV often developed encephalitis.

Clinical cases in birds

WEEV and EEEV infections seem to be asymptomatic in most birds residing in endemic areas; however, outbreaks or sporadic clinical cases have been reported occasionally. Many of the birds affected by EEEV had neurological signs ranging from tremors and incoordination to paresis or paralysis, but some had nonspecific signs alone. Some also developed gastrointestinal signs, including profuse diarrhea in some EEEV-infected pheasants; intermittent vomiting, followed by persistent regurgitation and diarrhea, in a captive colony of African penguins; and hemorrhagic enteritis with regurgitation and diarrhea in ratites. While most affected birds died or were euthanized, the majority of the African penguins recovered with intensive supportive care, with sequelae limited to subtle ataxia in some birds. WEEV has been reported to cause mild to severe illness in emus, with watery diarrhea or hemorrhagic enteritis, neurological signs and sudden death. It can also cause decreased egg production and reduced egg quality in turkeys.

Fort Morgan virus infections can result in encephalitis and hepatitis in house sparrow nestlings, while highlands J virus caused fatal illnesses in experimentally infected young chickens, turkeys and partridges and nonspecific signs of illness with decreased egg production in adult turkeys.

Post Mortem Lesions

Gross lesions from EEEV, WEEV and VEEV in equids are generally limited to the CNS, and can include meningeal congestion, darkened areas of necrosis and/or hemorrhages in the brain and spinal cord. Some animals may have no obvious macroscopic lesions at the time of death. Necrotic foci may be found occasionally in the pancreas, liver and/or heart. There can also be secondary lesions from antemortem trauma, dehydration or inappetence, and prolonged recumbency can result in pneumonia, particularly in foals.

Birds infected with EEEV can have both CNS and visceral lesions, including coelomic effusions, an enlarged, friable spleen and liver, swollen kidneys, necrohemorrhagic enteritis, and petechiae and ecchymoses on the serosa of various internal organs.

Diagnostic Tests

Clinical cases caused by EEEV, WEEV or VEEV in equids can be diagnosed with a positive IgM antibody-capture ELISA or a fourfold rise in antibody titers in a virus neutralization assay (e.g., the plaque reduction neutralization test). A single high titer in the latter test can help support a presumptive diagnosis. Other serological tests, such as hemagglutination inhibition and complement fixation, can also be used, though complement fixing antibodies tend to appear late. Vaccination history must be considered when interpreting antibody titers, and cross-reactivity can be an issue, particularly in some tests. Paired serum samples taken from nearby febrile animals can sometimes be helpful, as antibodies can begin to rise before the onset of neurological signs and a fourfold increase in titer my not be seen in the affected animal.

Live viruses, viral nucleic acids and/or antigens may be found in the brain of equids at necropsy, using virus isolation, RT-PCR or immunohistochemistry, respectively. EEEV is usually recovered more readily from the CNS than WEEV or VEEV. Viruses can also be detected occasionally in extracranial tissues such as the liver, spleen or pancreas, though this is not reliable. Some viruses, particularly the epidemic strains of VEEV, may be isolated from the blood of live animals or detected by RT-PCR during the early, febrile stage of the illness; however, viremia has usually disappeared by the time the neurological signs appear.

EEEV, WEEV and VEEV can be isolated in a number of vertebrate and mosquito cell lines, such as Vero, RK–13 or BHK–21 cells, as well as in embryonated eggs and, if necessary, in neonatal rodents (e.g., mice, hamsters) or chicks. The recovered virus can be identified with genetic techniques (e.g., PCR), serology or immunofluorescence. VEE viruses can be subtyped at a reference laboratory with tests such as immunofluorescence, differential PRN tests and nucleic acid sequencing. Distinguishing EEEV from Madariaga virus has generally required sending the sample to a reference laboratory as well; however, a RT-PCR assay reported to distinguish these two viruses has been published.

Similar tests can be used to diagnose infections in other mammals or birds.

Treatment

Treatment of affected animals is generally limited to supportive care. Many equids with severe encephalitis must be euthanized, as it can be difficult to treat these cases without risk of serious injury to the animal and personnel.

Control

Disease reporting

Veterinarians who encounter or suspect EEE, WEE or VEE should follow their national and/or local guidelines for disease reporting. VEEV (subtype I) is exotic to the U.S. and must be reported immediately to state or federal authorities. State reportable disease lists should be consulted for specific requirements for the other viruses.

Prevention

Equids are generally protected from EEE, WEE and epidemic VEE by vaccination. Vaccines are also administered sometimes to susceptible birds. Preventing transmission from mosquitoes is difficult, but housing

animals in screened barns, particularly during the hours of high mosquito activity, and other measures (e.g., mosquito repellents, fans, mosquito abatement measures) may reduce the number of bites. Movement controls on equids can help control epidemic VEE, as these animals are the primary amplifying hosts for these viruses.

Morbidity and Mortality

Eastern and western equine encephalomyelitis

EEEV and WEEV usually cycle inapparently in their wild hosts, only emerging occasionally to affect domestic animals. Extensive outbreaks, such as one WEE epidemic that affected more than 350,000 North American horses and mules in 1937-38, or a 1947 EEE outbreak that killed an estimated 12,000 horses in Louisiana, have not been seen since vaccines became available; however, sporadic cases or outbreaks still occur, particularly in unvaccinated horses or in the southern U.S., where the long mosquito season may outlast the duration of immunity from vaccination.

Clinical cases of EEE and WEE in equids may be seen year-round or seasonally, depending on the area. In temperate regions, they tend to peak in late summer and fall. The number of cases fluctuates from year to year, and may also show longer term patterns, though the reasons are unclear. Relatively few cases of WEE have been reported recently in North America; however, this virus caused outbreaks in Argentina and Uruguay in 2023/ 2024 after a prolonged absence. Conversely, some authors have suggested that EEE cases in North America might be rising.

WEE is generally less severe than EEE in equids, with a higher proportion of mild cases and subclinical infections. The estimated case fatality rate in most WEE outbreaks is around 20-30%, whereas 75-90% of horses with encephalitis caused by EEEV die or are euthanized, and many surviving animals have severe residual neurological signs. Madariaga virus outbreaks can also be severe. One outbreak that affected approximately 200 horses in Brazil had a case fatality rate of 73%. What percentage of EEEV infections is subclinical is uncertain, though two studies found antibodies to this virus in 2-9% of healthy unvaccinated horses. Clinical cases in other mammals seem to be infrequent, and might disproportionately affect young animals. Nursing piglets were the most severely affected age group during outbreaks in pigs, and most of the cases in dogs and wolves were in pups. The majority of EEE cases reported in non-equid hosts have been fatal, but it is possible that milder cases are missed.

Birds in endemic areas generally seem to be unaffected by EEEV or WEEV; however, it is possible that these viruses might have more severe effects if introduced to naive avian populations. Reported case fatality rates from EEEV in affected flocks or exhibits varied from 5% to > 80%, while the morbidity rate in flocks of WEEV-infected emus ranged from 15% to 50%, and approximately 9% of the birds died.

Venezuelan equine encephalomyelitis

Epidemic VEE viruses appear only sporadically, but can spread widely and cause epidemics that may last for several years. Up to 90% of susceptible equids may become infected, with morbidity rates that vary from 10-40% in some areas to 50-100% in others, and a case fatality rate of 20-90%. The case fatality rate was 30-50% in outbreaks caused by enzootic I-E variants in Mexico in the 1990s.

Infections in Humans

Incubation Period

The incubation period for EEE, WEE and VEE viruses is usually around 2-10 days.

Clinical Signs

Eastern and western equine encephalitis

WEEV, EEEV and Madariaga virus infections have a wide range of outcomes in people, from subclinical infections to severe encephalitis.

EEE usually begins abruptly, as a flu-like syndrome that can include fever, chills, headache, myalgia, arthralgia and abdominal pain, which may be severe enough to mimic an acute abdominal emergency. Vomiting and diarrhea may also be seen, and children with EEE sometimes develop generalized, facial or periorbital edema. Febrile illnesses caused by Madariaga virus in children may resemble dengue and have sometimes included a cough, rash, conjunctivitis and/or tonsilitis. Some patients infected with either virus recover completely after 1-2 weeks, while others develop neurological signs, sometimes after a period of apparent recovery. Encephalitis caused by EEEV is often severe and can include headache, altered mentation, focal neurological deficits, tremors, seizures and paresis or paralysis, sometimes progressing to coma. Death is common, and many survivors have permanent brain damage. While Madariaga virus infections tend to be milder, this virus can also cause severe encephalitis, persistent CNS deficits and deaths.

WEE is similar to EEE but often milder. An initial febrile, flu-like illness, which may occasionally include respiratory signs, is sometimes followed by neurological signs such as restlessness, irritability, tremor and signs of focal meningeal irritation or, infrequently, by more severe neurological signs that resemble EEE. CNS signs are more likely to occur in children, especially infants under a year of age, and are uncommon in healthy adults. Infants who recover may have severe CNS deficits, but permanent sequelae in children older than a year are usually limited to persistent seizures if there were convulsions during the illness. Most adults recover completely.

Venezuelan equine encephalitis

The symptoms caused by endemic and epidemic VEE viruses in humans are similar. The most common syndrome is an acute flu-like illness with nonspecific clinical signs that may include fever, chills, generalized malaise, severe headache, photophobia, and myalgia particularly in the legs and lower back. Coughing, sore throat, nausea, vomiting and diarrhea may also be seen, and other signs, such as a macular rash or arthralgia in the wrists and ankles, have been reported

in some epidemics. Reproductive losses including abortions, stillbirths, fetal encephalitis and congenital neurological anomalies are also possible. Mild to severe neurological signs can be seen in a small percentage of affected children, and to a lesser extent in adults over the age of 50 years, but are usually absent in other adults. Deaths are rare.

Diagnostic Tests

Clinical cases caused by EEEV, WEEV and VEEV are often diagnosed serologically by conversion from IgM to IgG, a fourfold increase in antibody titers, or the detection of specific IgM in cerebrospinal fluid (CSF). A single high antibody titer may be suggestive. Viruses and/or their nucleic acids can sometimes be found in the blood during the early flu-like stage of the illness, and VEEV (and rarely other viruses) may be detectable in throat swabs. Viruses have usually disappeared from the blood before the onset of encephalitis; however, they can sometimes be found in CSF, or in CNS tissues at autopsy. Some viruses can be detected more readily than others, with WEEV reported as being particularly difficult to find.

Treatment

Treatment consists mainly of supportive and symptomatic care.

Control

Measures to prevent mosquito bites, including avoidance of mosquito habitats and periods of peak activity, and the use of repellants and protective clothing (e.g., mesh outerwear, long pants, long-sleeved shirts) can reduce the risk of infection. Surveillance programs in birds and reports of cases in equids help predict human outbreaks and can be useful for targeting protective measures, including the use of mosquito abatement programs, to high risk periods. Because equids amplify epidemic VEE viruses, controlling these viruses in equids is also helpful in reducing the risk to humans.

Containment level 3 is required for work with EEEV, WEEV or VEEV in the laboratory. Investigational vaccines have sometimes been made available for certain people at high risk of infection with these viruses, such as laboratory workers. Precautions should also be taken to prevent exposure to body fluids when performing necropsies on potentially infected animals.

Morbidity and Mortality

Eastern and western equine encephalitis

Eastern and Western encephalitis outbreaks in people usually parallel those in equids, occurring mainly in late summer to fall in temperate regions, but year-round in some warmer areas, with case numbers fluctuating from year to year. In the U.S., where mandatory reporting has been in place since 2002, the annual incidence of clinical EEE is about 0.03 cases per million population. On average, 5-10 clinical cases are reported most years; however, this number varies from zero to around 30-40, though the latter is unusual (e.g., 32 cases in 1959, 38 in 2019). How often Madariaga virus affects people in South America is unclear, but a recent report of 8 mild cases in children, identified during routine surveillance of influenza-like illnesses at a Caribbean school, suggests that infections might be relatively common in some regions.

Encephalitis caused by EEEV can occur in all age groups, though it is most common in those over 60 years of age or younger than 15. Estimates of the case fatality rate range from 30% to 75%, and are currently around 30-40% in the U.S. Permanent neurological deficits are common in survivors. The incidence of mild or subclinical EEEV infections is uncertain; however, relatively mild cases have been found accidentally during surveillance for other diseases, and serological surveys after some outbreaks suggested that approximately 4-5% of infected people become ill. While Madariaga virus can also cause severe encephalitis, clinical cases caused by this virus seem to be overall less severe, and some case series describing small numbers of hospitalized patients reported no deaths.

WEE outbreaks have been documented infrequently in South America; however, this disease was relatively common in North America at one time. An average of 34 confirmed cases (range 0 to 172.) occurred annually in the U.S. between 1955 and 1984, and there are historical reports of more extensive epidemics, including one in 1941 that affected more than 3000 people in the U.S. and Canada. For reasons that are unclear, this disease seems to have become significantly less common in North America in recent decades. Clinical cases of WEE tend to be relatively mild in healthy adults, though they can be more severe in the elderly and children, particularly infants. The estimated case fatality rate is around 3-4%, with a high of 8-15% during the 1941 epidemic. Sequelae are most severe in infants, with approximately half of infants under a month of age developing permanent neurological damage

Venezuelan equine encephalomyelitis

VEE epidemics can affect large numbers of people, with more than 10% of the population sometimes becoming ill. Serological studies suggest that enzootic VEE viruses might also cause significant numbers of clinical cases in Latin America, but may be mistaken for other diseases such as dengue. Most infections with either epidemic or enzootic VEE viruses in healthy adults are mild or asymptomatic, with an estimated case fatality rate of $\leq$ 1%. However, symptomatic cases can have a 4-15% incidence of mild to severe neurological signs in very young or elderly patients. The case fatality rate in patients who develop encephalitis is estimated to be 10-35%, with the highest rates in young children.

Internet Resources

eMedicine. Eastern equine encephalitis

eMedicine. Western equine encephalitis

Public Health Agency of Canada. Pathogen Safety Data Sheets

The Merck Manual

The Merck Veterinary Manual

United States Department of Agriculture. Animal and Plant Health Inspection Service. Equine encephalitis (EEE/WEE/VEE)

U.S. Centers for Disease Control and Prevention (CDC). Eastern equine encephalitis

World Organization for Animal Health (WOAH)

WOAH Manual of Diagnostic Tests and Vaccines for Terrestrial Animals

WOAH Terrestrial Animal Health Code

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