Etiology

Wesselsbron disease is caused by Wesselsbron virus, an arthropod-borne virus in the genus *Flavivirus* of the family *Flaviviridae*. This virus has the properties typically found in a hemagglutinating flavivirus, but is not yet well-characterized.

Geographic Distribution

Wesselsbron virus has been isolated from vertebrates or arthropod vectors in several countries in southern Africa including Cameroon, the Central African Republic, Nigeria, Senegal, South Africa, Uganda, and Zimbabwe. There is serological evidence of its presence in other sub-Saharan countries in Africa including the island of Madagascar. In addition, this virus has been isolated from mosquitoes in Thailand.

Transmission

Wesselsbron virus is transmitted by mosquitoes in the genus *Aedes* including *A. caballus* and *A. circumluteolus*. A high seroprevalence among domestic herbivores in warm, moist regions suggests that these animals may have a role as viral reservoirs. In ruminants, there seems to be no direct virus transmission between animals; however, humans have been infected by handling the virus or contaminated material; in these cases, transmission was thought to occur by contact or possibly aerosols.

Disinfection

Although there are no studies describing the disinfectant sensitivity of Wesselsbron virus, this virus has the properties typical of hemagglutinating flaviviruses. These viruses are sensitive to temperatures above 40°C (104°F), detergents, and lipid solvents. The Wesselsbron virus is relatively fragile and is unlikely to remain infectious for more than a few days in carcasses or animal products in the field.

Infections in Humans

Incubation Period

The incubation period in humans is 2 to 4 days.

Clinical Signs

Most infections in humans seem to be subclinical or consist of mild symptoms, such as fever, that are not investigated. In the few symptomatic cases that have been reported, Wesselsbron disease has resembled influenza. The symptoms included fever, headache, arthralgia, and muscle pains. Cutaneous hypersensitivity and mild skin rashes have also been reported. Although the fever usually disappears after 2-3 days, the muscle pains have been known to persist for much longer.

Communicability

There are no reports of person-to-person transmission with Wesselsbron virus.

Diagnostic Tests

Wesselsbron virus has been isolated from the blood or serum of febrile patients, as well as from a pharyngeal wash. Antibodies in humans have been detected by hemagglutination inhibition tests.

Treatment

Treatment is symptomatic; there is no specific treatment for the virus.

Prevention

People who work with Wesselsbron virus or contaminated tissues should wear gloves and other protective clothing, and avoid techniques that would aerosolize the virus. Mosquito control measures such as repellants and mosquito netting also decrease the risk of infection.
Morbidity and Mortality

Subclinical infections are common among people in parts of southern Africa. Clinical infections have rarely been reported; in most cases, they were seen in laboratories or among field personnel who handled the virus or contaminated material. Fatal infections have not been reported in humans.

Infections in Animals

Species Affected

Wesselsbron disease is seen mainly in sheep. Goats, cattle, pigs, and horses can also be infected; a link with abortions is suspected in cattle and goats.

The reservoir host is unknown, but may include domestic ruminants, wild birds, wild rodents such as gerbils, or other animals.

Sporadic infections have been reported from other species. Infections have reported in ostriches, but their significance as a host is unclear. The virus has also been recovered from a camel and a fatal infection in a dog. Antibodies have been found in lemurs in Madagascar.

Incubation Period

The incubation period in sheep is 1 to 4 days.

Clinical Signs

Sheep

Most clinical cases are reported in sheep, particularly pregnant ewes and newborn lambs. Abortions are the usual symptom in pregnant ewes. They are often accompanied by neurologic defects in the fetus and sometimes by hydrops amnii (excessive accumulation of amniotic fluid in the amniotic cavity). Complications of the abortion may lead to the death of the ewe.

In newborn lambs, the clinical signs may include fever, anorexia, weakness, and an increased respiratory rate. Up to 30% of affected lambs may die.

In non-pregnant adult sheep, the disease usually is limited to fever; however, during one atypical outbreak in South Africa in 1957, the symptoms also included a nasal discharge, diarrhea, jaundice, and subcutaneous edema of the head. Many of the sheep died in this epidemic. Although it is not known whether concurrent infections or other factors were present, it is possible that the virus combined with chronic copper poisoning to trigger this condition.

Other species

Infections with Wesselsbron virus in adult goats, cattle, horses, and pigs are usually inapparent and limited to fever. The virus has been associated with abortions in goats in Botswana. It is also suspected as a cause of abortion in cattle. However, the incidence of abortion in this species may be low; in one experiment, infection of 15 pregnant cows resulted in no abortions. A fatal infection has been reported in a dog.

Communicability

The viremia in sheep and cattle is high enough to infect mosquitoes; however, there does not seem to be any direct animal-to-animal transmission in ruminants.

There are no published reports on the persistence of the virus in meat, but humans have been infected by handling carcasses during necropsies. There are no reports studying the transmission of this virus in milk, semen, embryos, or feces.

Diagnostic Tests

Wesselsbron disease can be diagnosed by virus isolation or serology.

The Wesselsbron virus can be isolated from most organs of lambs that die during the clinical stage. Commonly used tissues include the blood, serum, liver or brain from aborted fetuses, and the spleen or liver from dead lambs. The virus is isolated by inoculation into suckling mice or chick embryos, or tissue culture in BHK or lamb kidney cells. The identity of the virus can be confirmed by virus neutralization.

Immunohistochemistry, using formalin-fixed liver tissues, has been used to confirm the disease in newborn lambs.

Serologic tests include hemagglutination-inhibition, complement fixation, and virus neutralization. Although there is a high degree of cross-reactivity with other flaviviruses in hemagglutination-inhibition tests, the homologous Wesselsbron titers are usually much higher than the heterologous titers. An ELISA has also been published; this test had greater sensitivity and lower cross-reactivity than the hemagglutination inhibition test.

Treatment

Treatment is supportive and symptomatic; there is no specific treatment for the virus.

Prevention

Sheep can be immunized with an attenuated vaccine. Immunity is lifelong. Although vector control is also theoretically possible, it is usually not practical.

Morbidity and Mortality

The infection rate appears to be high in ruminants in endemic regions of Africa. In cattle, sheep, and goats, the seroprevalence may be as high as 50%. Animals of all ages seem to be susceptible to infection. Infections are seen year-round in some regions. In others, outbreaks mainly occur when heavy rains favor the replication of mosquitoes that breed in floodwaters.

The mortality rate in newborn lambs can be as high as 30%. Epizootics have occurred in sheep in southern Africa.
Most infections in adult animals are subclinical and nonfatal.

**Post-Mortem Lesions**

Congenital malformations of the CNS and arthrogryposis have been reported in ovine and bovine fetuses. Necropsy lesions in young lambs may include hepatomegaly with a yellowish to orange-brown liver, and moderate to severe icterus. Petechiae and ecchymoses are common in the abomasal mucosa; the contents of the abomasum may be chocolate-brown from gastrointestinal hemorrhages. Subcutaneous edema has also been reported. On histopathology, there is usually mild to extensive necrosis of the liver parenchyma, with foci of necrotic hepatocytes. Lesions in adult animals are similar but usually much milder.

**Internet Resources**

The Merck Veterinary Manual

http://www.merckvetmanual.com/mvm/index.jsp

Universal Virus Database


**References**


