

Getah Virus Infection

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Importance

Getah virus is a mosquito-borne virus that can cause a mild, self-limiting illness in horses. This virus is widely distributed from Eurasia to Australasia, and antibodies have been found in many species of animals. However, clinical disease seems to be very rare. The first known outbreak occurred in Japan in 1978; it affected racehorses at two densely populated training stables. A small number of cases was also reported in Japanese horses in 1983. In 1990, clinical disease was reported outside Japan, at a thoroughbred breeding farm in India. In addition, Getah virus is suspected of being pathogenic in pigs.

Etiology

Getah virus is a member of the Getah subgroup in the genus *Alphavirus* and family *Togaviridae*. There are at least eight strains of this virus, which appears to mutate fairly often. Sagiya virus, which is found in Japan and causes identical symptoms in horses, is very closely related to Getah virus. Some researchers consider Sagiya virus to be a strain of Getah virus.

Species Affected

Clinical signs have been reported only in horses, pigs, and experimentally infected mice. However, serologic studies suggest that asymptomatic Getah virus infections occur in many vertebrates including horses, pigs, cattle, water buffalo, kangaroos, wallabies, birds, reptiles, non-human primates, and humans. Mice, rats, rabbits, hamsters, and guinea pigs can be infected experimentally.

Geographic Distribution

Serologic studies suggest that Getah virus is widely distributed from Eurasia (including Siberia and Mongolia) to Australasia. As of January 2006, clinical disease had been reported only in Japan and India.

Transmission

Getah virus appears to be maintained in a natural cycle between mosquitoes and various vertebrate hosts. This virus seems to be transmitted mainly by various species of *Aedes* and *Culex*; the specific vector varies with the climate and geographic region. The amplifying hosts are thought to include horses, pigs, and possibly other species such as rodents.

Getah virus is also directly contagious between horses, probably via aerosols or direct contact with nasal secretions. This route is thought to be uncommon; high doses of virus are needed to establish intranasal infections, but only small amounts of virus are found in the nasal secretions of naturally infected horses. Getah virus has not been recovered from the feces or urine of horses; however, it was found in the feces of an experimentally infected piglet with diarrhea.

Vertical transmission has been reported in pigs, as well as in experimentally infected mice, hamsters, guinea pigs, and rabbits. There is no indication that vertical transmission occurs in horses.

Incubation Period

The incubation period in both horses and pigs is short. Horses infected experimentally by the intranasal route become symptomatic in 3 to 4 days. After intramuscular injection, the incubation period is 2 to 6 days. Experimentally infected pigs become symptomatic 1 to 3 days post-inoculation.

Clinical signs

In horses, Getah virus infection is a mild, self-limiting illness characterized by fever, hind limb edema mainly in the fetlock, and stiffness. Swelling of the submandibular lymph nodes, mild abdominal pain, depression, mild icterus, and scrotal edema have also been reported. An urticarial rash is occasionally seen. This rash consists of 3-5 mm papules mainly on the neck from the shoulder to the forearm, and over the hindquarters from the thigh to the gaskin. Serous nasal discharge has been reported in experimentally infected horses but not in natural cases. In some horses,

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the clinical signs are limited to one or two symptoms such as fever alone, fever and a skin rash, or fever and limb edema. Getah virus does not seem to cause abortions or birth defects in horses; pregnant mares delivered normal foals after an outbreak at a breeding farm in India. Horses recover fully in approximately 1 to 2 weeks.

Getah virus may also be pathogenic in swine. In a few cases, fetal deaths have been reported after natural or experimental infection of sows. Other symptoms reported in experimentally infected pigs include transient fever, anorexia and, in young piglets, mild depression and diarrhea.

Experimentally infected neonatal mice develop polymyositis. Fetal deaths and decreased litter sizes have been reported in pregnant mice.

Post Mortem Lesions

Deaths have not been reported in naturally infected horses. Generalized lymphadenopathy was reported in experimentally infected horses; the splenic and inguinal lymph nodes were particularly prominent. Some horses also had a slightly enlarged spleen and/or liver. Moderate glomerular congestion was noted in one horse, while another had congestion and slight turbidity of the pia mater in the brain. In horses with a rash, the subcutaneous tissues were moderately edematous due to lymph stasis. These horses had scattered maculae in the dermis; on cut section, the maculae were thickened and circumscribed, with pale reddish foci.

The histopathologic lesions consisted mainly of moderate lymphoid hyperplasia in the submandibular, axillary, splenic, renal and inguinal lymph nodes, as well as in the spleen. The skin lesions contained perivascular or diffuse infiltrates of lymphoid cells and histiocytes, thickened blood vessel walls, and edematous smooth muscle cells. Scattered hemorrhagic foci and eosinophilic infiltrates were seen in some lesions. Perivascular cuffing with mononuclear cells was reported in the cerebrum of two horses.

Morbidity and Mortality

Although clinical disease in horses is rare, many horses are seropositive in endemic areas. In Japan, 3% to 50% of horses have antibodies to Getah virus; a few surveys have reported seroprevalence rates as high as 93%. Retrospective serologic studies suggest that the virus was widely distributed in Japan before the first known outbreak in 1978. The reason for the high exposure rate but paucity of clinical cases is unknown. It has been suggested that the passage of Getah virus through large groups of horses may result in increased virulence.

The morbidity and mortality rates are known only for two outbreaks. During a 1978 outbreak in Japan, the virus spread slowly and irregularly over a 4 to 6 week period at two training centers for racehorses. The morbidity rate was 40%. During a 1990 Indian outbreak, the infection spread rapidly, and all cases appeared over a period of 12

days. In this outbreak, the morbidity rate was 30%. The symptoms of Getah virus infection are transient and mild, and deaths have not been reported.

Experimental infections suggest that Getah virus is mildly pathogenic for swine. Deaths have not been reported in young or adult animals, but the virus may be responsible for some fetal deaths.

Diagnosis

Clinical

In horses, Getah virus infection is suggested by a fever, hind limb edema, and stiffness. A rash or swelling of the submandibular lymph nodes also supports the diagnosis. The clinical diagnosis should always be confirmed by laboratory testing. In pigs, Getah virus infection may be a consideration in cases of reproductive disease; however, this link has not been conclusively established.

Differential diagnosis

The differential diagnosis includes equine viral arteritis and the mild form of African horse sickness (horsesickness fever).

Laboratory tests

Getah virus can be isolated in VERO, RK-13, BHK-21, and many other cell lines as well as by intracerebral inoculation of suckling mice. In horses, serologic tests include serum neutralization, complement fixation, and hemagglutination inhibition. An enzyme-linked immunosorbent assay (ELISA) test has been developed for swine.

Samples to collect

Before collecting or sending any samples from animals with a suspected foreign animal disease, the proper authorities should be contacted. Samples should only be sent under secure conditions and to authorized laboratories to prevent the spread of the disease.

In horses, plasma collected at the onset of pyrexia is preferred for virus isolation; viremia appears to occur only during the first 1 to 2 days after the onset of fever. Getah virus has also been isolated from saliva, nasal swabs, and defibrinated blood. In addition, virus isolation may be attempted from the lungs, liver, spleen, kidneys, lymph nodes, and spinal cord at necropsy. In experimentally infected horses, the axillary and inguinal lymph nodes have the highest viral titers and seem to contain virus for the longest period.

The virus has been isolated from pigs at 1 to 2 days post-inoculation; the virus was found in the spleen, lymph nodes, and feces. It has also been isolated from dead porcine fetuses.

In both species, paired acute and convalescent samples can be collected for serology; due to the high prevalence of seropositive animals, single serum samples are less useful.

Recommended actions if Getah virus infection is suspected

Notification of authorities

Getah virus infection should be reported immediately to state or federal authorities upon diagnosis or suspicion of the disease.

Control

Getah virus appears to be contagious to some extent. The disinfectant susceptibility of this virus has not been published; however, Togaviruses are not very stable in the environment and can be inactivated by most disinfectants. Two related Alphaviruses, the Eastern and Western equine encephalitis viruses, are susceptible to 1% sodium hypochlorite, 2% glutaraldehyde, formaldehyde, and 70% ethanol, as well as moist and dry heat.

Vector control measures may also help prevent transmission, as Getah virus seems to be spread mainly by mosquitoes.

An inactivated vaccine is available for horses in Japan. Vaccinated horses are protected from both clinical disease and viremia. In Japan, most 2-year old racehorses in training receive two doses of the vaccine, followed by a booster each year.

Public Health

Although antibodies to Getah virus have been found in humans, there are no reports of symptomatic infections.

Internet Resources

International Veterinary Information Service (IVIS)
<http://www.ivis.org>

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