

Getah Virus Infection

Last Updated: March 2017

Importance

Getah virus is a mosquito-borne pathogen that can cause a mild, self-limited illness in horses, and might also cause reproductive losses in pigs. This virus is widely distributed from Eurasia to Australasia, and antibodies have been found in many animal species. However, clinical cases have been documented mainly during a few outbreaks among racehorses at training centers in Japan, and an outbreak at a Thoroughbred breeding farm in India in 1990. Racehorses in Japan are now vaccinated during training, and no cases were reported there between 1984 and 2014. However, some animals were again affected at a Japanese training facility in 2014 and 2015. Most, but not all, cases occurred in young horses that had begun, but not yet completed, their initial vaccinations. What causes Getah virus to emerge and cause outbreaks occasionally, while circulating subclinically in horse populations at other times, is still unclear.

Etiology

Getah virus is a member of the Getah subgroup in the genus *Alphavirus* and family *Togaviridae*. There are a number of viral strains. Sagiya virus, originally found in pigs, is now considered to be a strain of Getah virus.

Species Affected

Clinical signs have been reported only in horses, pigs and experimentally infected mice. However, serological studies suggest that asymptomatic Getah virus infections may occur in many vertebrates including mammals (e.g., horses, pigs, wild boar, cattle, water buffalo, non-human primates), marsupials (kangaroos, wallabies), birds and reptiles. Mice, rats, rabbits, hamsters and guinea pigs have been infected experimentally.

The maintenance hosts are unknown, but both horses and pigs can develop viremia sufficient to infect mosquitoes.

Zoonotic potential

Although antibodies to Getah virus have been found in humans, there are no reports of any illnesses associated with this virus.

Geographic Distribution

Getah virus has been isolated in a number of Asian countries, and serological studies suggest that it is widely distributed from Eurasia (including Siberia and Mongolia) to Australasia. Japan and India are the only countries that have published descriptions of clinical cases; however, the illness is mild and could be confused with other diseases.

Transmission

Getah virus appears to be maintained in a natural cycle between mosquitoes, especially members of the genera *Aedes* and *Culex*, and various vertebrate hosts. *C. tritaeniorhynchus* and *A. vexans* are thought to be the most important vectors in Japan and South Korea. Amplifying hosts may include horses, pigs and possibly other species such as rodents.

Getah virus can also be transmitted directly between horses, probably via aerosols or by direct contact with nasal secretions. This route is thought to be uncommon; high viral doses are necessary to establish intranasal infections, but only small amounts of virus have been 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 and experimentally infected mice, hamsters, guinea pigs and rabbits. There is no indication that vertical transmission occurs in horses.



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Disinfection

The disinfectant susceptibility of Getah virus has not been published, but members of the *Togaviridae* can usually be inactivated by most disinfectants, including sodium hypochlorite and other oxidizing agents, sodium hydroxide, aldehydes (e.g., glutaraldehyde, formalin), 70% ethanol and detergents.

Incubation Period

The incubation period in both horses and pigs is short. Experimentally infected horses became symptomatic 3-4 days after intranasal inoculation, and 2-6 days after intramuscular injection. Experimentally infected pigs developed clinical signs in 1-3 days.

Clinical Signs

In horses, Getah virus causes a mild, self-limited illness characterized by fever, leg edema that typically affects the hindlimbs (especially the fetlock) and stiffness. Swelling of the submandibular lymph nodes, mild abdominal pain, depression, mild icterus and scrotal edema have also been reported. Horses occasionally develop an urticarial rash, with small papules that are usually most common on the neck from the shoulder to the foreleg, 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, there may be only one or two clinical signs, 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 within approximately 1-2 weeks.

Getah virus may also be pathogenic in swine. In a few cases, fetal deaths were reported after natural or experimental infection of sows. Some experimentally infected pigs developed transient fever, anorexia and mild depression, and some young piglets had diarrhea. Other experimentally infected pigs had no clinical signs.

Experimentally infected neonatal mice developed polymyositis. Fetal deaths and decreased litter sizes were reported in experimentally infected pregnant mice.

Post Mortem Lesions

Deaths have not been reported in naturally infected horses. Experimentally infected horses had generalized lymphadenopathy, with the splenic and inguinal lymph nodes especially 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 lymphatic stasis. These horses had scattered maculae in the dermis; on cut section, the maculae were thickened and circumscribed, with pale reddish foci.

The histopathological lesions consisted mainly of moderate lymphoid hyperplasia in the lymph nodes and

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.

Diagnostic Tests

Getah virus infections in horses can be diagnosed by isolating the virus or detecting viral nucleic acids with RT-PCR assays, using samples of blood (serum), saliva and nasal swabs. Viremia seems to occur only during the first 1-2 days after the onset of fever. In experimentally infected horses, this virus has also been found in a number of tissues including the lungs, liver, spleen, kidneys, lymph nodes and spinal cord, with the highest and most persistent viral titers in the axillary and inguinal lymph nodes. In experimentally infected pigs, Getah virus was isolated from the blood (serum), spleen, kidney, lymph nodes, tonsils and feces, 1-4 days after inoculation. It was also found in dead porcine fetuses. Oral and nasal swabs from pigs did not contain the virus in one study. 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.

Serological tests that may be used for diagnosis in horses include serum neutralization, hemagglutination inhibition and complement fixation. A rising titer in paired samples is diagnostic. An enzyme-linked immunosorbent assay (ELISA) test was developed for swine. Due to the high prevalence of seropositive horses and pigs in endemic regions, single serum samples are difficult to interpret.

Treatment

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

Control

Disease reporting

Veterinarians who encounter or suspect a Getah virus 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 of any livestock disease suspected to be exotic.

Prevention

Racehorses at training centers in Japan are usually vaccinated against Getah virus. Two-year-old horses receive two doses of an inactivated vaccine, followed by a booster each year. Serological studies suggest that this vaccine is effective against the viruses responsible for outbreaks in 2014 and 2015. Other horses can also be vaccinated; however, this seems to be uncommon. Control of the mosquito vectors could theoretically help prevent transmission, but adequate mosquito control may be difficult and impractical. Sick animals might transmit Getah virus to nearby animals, although this route appears uncommon, and basic infection control measures seem warranted.

Morbidity and Mortality

Clinical cases in horses are usually mild and self-limited, and deaths have not been reported. Only a few outbreaks have been described, although many horses are seropositive in endemic areas. In Japan, surveys have found antibodies to Getah virus in 3-50% of horses, with a few studies reporting seroprevalence rates as high as 93%. Illnesses were first described in Japan in 1978, although the virus was present before that time. During these outbreaks, Getah virus infections appeared over a 4-6 week period at two training centers for racehorses. The morbidity rate was 40%, while 34% of the animals seroconverted asymptotically. Smaller numbers of cases occurred at other training facilities in 1979 and 1983. The only outbreak described outside Japan was at a Thoroughbred breeding center in India in 1990. In this instance, the morbidity rate was 30%, and all cases appeared over a period of 12 days.

Racehorses in Japan were vaccinated after the initial outbreaks, and no additional cases were reported until 2014 and 2015, when outbreaks occurred at a training center previously affected in 1978. Cases were seen in September and October in 2014, and from late August through October in 2015. Although this center houses approximately 2,000 horses, only small numbers of animals became ill, with morbidity rates of approximately 1.5% to 3%. Additional horses (approximately 7% in 2014) seroconverted without clinical signs. Two-year-olds were disproportionately affected, comprising 61% of the clinical cases in 2014 and 90% in 2015. Most horses at this facility receive their first Getah virus vaccination upon entry, and horses that had only received a single dose of vaccine (of a 2-dose series) were more likely to become ill. Outside the training center, an increasing number of horses in the region became seropositive during this time, apparently without reports of illness.

It is unclear when and why Getah virus emerges to cause clinical cases. Cases seem to be rare or underdiagnosed in most equine populations, although vaccination is uncommon outside training facilities in Japan. Factors proposed to contribute to outbreaks include the introduction of strains with increased virulence and/or exposure to higher viral doses. The virus isolated in 2014 and 2015 did not appear to be significantly more virulent for horses than older strains circulating in Japan.

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

Internet Resources

[International Veterinary Information Service \(IVIS\)](#)

[World Organization for Animal Health \(WOAH\)](#)

Acknowledgements

This factsheet was written by Anna Rovid Spickler, DVM, PhD, Veterinary Specialist from the Center for Food Security and Public Health. The U.S. Department of Agriculture Animal and Plant Health Inspection Service (USDA APHIS) provided funding for this factsheet through a series of cooperative agreements related to the development of resources for initial accreditation training.

The following format can be used to cite this factsheet. Spickler, Anna Rovid. 2017. *Getah Virus Infection*. Retrieved from <http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.php>.

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