Aino Disease

Aino Virus Infection

Last Updated: March 2006

Importance

Aino virus is one of several closely related viruses that cause a fetal deformity syndrome, accompanied by stillbirths and premature births, in ruminants. This virus does not appear to cause clinical signs, other than abortions, in adults.

Etiology

Aino virus is a member of the Simbu serogroup of the genus Orthobunyavirus, family Bunyaviridae. It is closely related to the Akabane, Peaton, Douglas and Tinaroo viruses.

Species Affected

Aino virus has been associated with fetal deformities in cattle and sheep. Antibodies to this virus have also been found in goats, buffalo, wild ruminants, and humans.

Geographic Distribution

Aino virus is widely distributed in Asia and Australia. Clinical cases of Aino disease have been reported from Japan and Australia.

Transmission

Aino virus is transmitted between animals by insect vectors. It has been isolated from mosquitoes and Culicoides spp.

Incubation Period

Fetal defects caused by Aino virus are seen at term, or months after infection of the dam. The length of this period can be estimated from natural or experimental infections. In one Japanese outbreak, virus isolation was primarily in July to August 1995, and affected calves were born mainly in December to March 1996; this suggests that the fetuses were infected from 120 to 180 days of gestation. In an experimental study in cattle, the critical gestational age when infection resulted in congenital malformations appeared to be 132 to 156 days.

Clinical Signs

Aino virus has been associated with fetal deformities in cattle, sheep, and experimentally infected chicks. Clinical signs have not been reported in non-pregnant adults with the exception of one report of sudden astasia and leukopenia in a naturally infected dairy cow. In naturally infected pregnant cattle and sheep, Aino virus has been associated with stillbirths, premature births, and birth defects including arthrogryposis, scoliosis, sunken eyes, cataracts, maxillary retraction, and dental irregularities. Some calves may have a domed head from hydranencephaly and cerebellar hypoplasia. Surviving calves may be weak and can have difficulty suckling or standing. They may also be blind or have poor eyesight. In addition, they may display a variety of neurologic signs including ataxia, torticollis, tetany, paresis, swimming movements, opisthotonus, and circular walking.

The first report describing experimental infections in pregnant cattle was published in 2004. Intravenous inoculation of five pregnant cows caused no symptoms other than a transient fever, and all five calves were normal. Intrauterine inoculation of five cows resulted in polyhydramnios, premature births, and deformities. Four cows gave birth to stillborn calves, while the fifth calf was born alive but weak. This calf had astasia and did not suckle. Arthrogryposis, scoliosis, hydranencephaly, and cerebellar hypoplasia were reported in the calves. Aino disease has not yet been reproduced by virus inoculation into pregnant ewes.

Aino virus is also teratogenic in experimentally infected chick embryos; in this species, it causes hydranencephaly, cerebellar hypoplasia, scoliosis, and arthrogryposis.
Post Mortem Lesions

Polyhydramnios is the only lesion that has been reported in adults; it was seen in cows infected by intrauterine inoculation. Affected calves may be stillborn or premature. Affected calves may be stillborn or premature. The typical birth defects associated with Aino virus infection are arthrogryposis, hydranencephaly, and cerebellar hypoplasia or agenesis. Other reported defects including necrotic foci in the cerebrum, cerebral cysts, micrencephaly, holencephaly, partial absence of the cerebrum, and hydrocephalus. In addition, there may be scoliosis, torticollis, maxillary retraction, sunken eyes, cataracts, and dental irregularities.

Histopathologic lesions may include gliosis, cerebellar cortical dysplasia, dilation of the mesencephalic aqueducts, loss of the cerebral parenchyma as a result of ventricular dilation, perivascular cuffing, proliferation of the blood vessels, and the presence of thick-walled blood vessels in the brainstem. The number of neurons in the ventral horn of the spinal cord may be decreased, and there may be myodysplasia of the skeletal muscle.

Morbidity and Mortality

Aino disease is seasonal; however, fetal defects occur some time after the activity of the insect vectors rather than coinciding with this period. In one outbreak in Japan, affected calves were born mainly from November to March. Both sporadic infections and severe outbreaks involving as many as 2000 calves have been reported.

The morbidity rate is undetermined. One study suggests that the fetal infection rate may be low; all of the calves from five intravenously inoculated cattle were normal. However, in one large outbreak, 43% of infected cows gave birth prematurely. The mortality rate is high in affected calves.

Diagnosis

Clinical

Aino disease should be suspected when premature or stillborn fetuses are born with arthrogryposis, hydranencephaly, and cerebellar hypoplasia. No history of disease is expected in the dam.

Differential diagnosis

Stillbirths, premature births, and fetal deformities can be caused by a variety of nutritional factors and toxins. Infectious diseases in the differential diagnosis include bluetongue, Akabane, Cache Valley disease, Chuzan virus infection, bovine virus diarrhea, border disease, and Wesselsbron disease.

Laboratory tests

Aino disease is usually diagnosed by serology and histopathology. Most often, the diagnosis is made by the presence of neutralizing antibodies in precolostral serum from the calf, or rising titers in the dam. Neutralizing antibodies are usually determined by the microtiter method. The Aino virus, akabane virus and other Simbu group viruses can cross-react in serologic tests.

Techniques to detect the Aino virus or its antigens are also available. Immunohistochemistry may find viral antigens in neuroglial cells in the brain. A nested RT-PCR assay can differentiate Aino disease from akabane. Virus isolation is also possible, but it is an uncommon method of diagnosis.

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.

Serum or cerebrospinal fluid can be collected from the fetus for serology. Precolostral serum samples should be taken from affected calves. Paired serum samples are the best way to demonstrate infections in the dam; however, by the time the clinical signs appear, rising titers may not be detected. Brain samples from the fetus or calf can be used for immunohistochemistry as well as histopathology.

Recommended actions if Aino disease is suspected

Notification of authorities

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

Federal: Area Veterinarians in Charge (AVIC):
www.aphis.usda.gov/animal_health/area_offices/

State Veterinarians:
www.usaha.org/Portals/6/StateAnimalHealthOfficials.pdf

Control

Aino virus does not seem to be transmitted between animals except by insect vectors. Care should be taken to prevent infection by potential vectors such as mosquitoes or gnats. If disinfection is necessary, enveloped viruses such as the Bunyaviridae are susceptible to most common viral disinfectants including hypochlorite (bleach), detergents, chlorhexidine, alcohol, phenols, and commercial disinfectants.

An inactivated vaccine has been developed and is being used in Japan.

Public Health

Antibodies to Aino virus have been found in humans but there are no reports of human disease. It is possible that these antibodies represent a cross-reaction to other bunyaviruses.
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* link defunct as of July 2012