Porcine Rubulavirus Infection

La-Piedad-Michoacan Paramyxovirus Infection, Blue Eye Disease

Last Updated: March 2006

Importance
Porcine rubulavirus infection or “blue eye” disease is an emerging disease first seen in La Piedad, Michoacan, Mexico and the neighboring states of Jalisco and Guanajuato in 1980. It is characterized by encephalitis and respiratory disease in piglets, reproductive failure in adult pigs, and occasional corneal opacity in all ages.

Etiology
Blue eye disease is caused by the porcine rubulavirus, which is also called the La-Piedad-Michoacan paramyxovirus (LPMV). This virus, which was first isolated in Mexico in the early 1980s, belongs to the genus Rubulavirus and family Paramyxoviridae. Only one serotype is known.

Species Affected
Pigs are the only known host species.

Geographic Distribution
Blue eye disease has been reported only from Mexico; however, closely related paramyxoviruses of pigs have been found in other countries including Australia, Canada, Japan, and Israel.

Transmission
Infections seem to be spread mainly by the respiratory route. A large amount of infectious virus has also been found in the urine. Vertical transmission occurs in utero.

Incubation Period
In experimental studies, symptoms appeared 3 to 5 days after intranasal inoculation of piglets.

Clinical Signs
In 2 to 21 day old suckling pigs, blue eye disease is characterized by encephalitis, pneumonia, and corneal opacity. Typically, the disease begins with the sudden onset of fever, arched back, and prostration or depression. These symptoms are followed by progressive neurologic disease with weakness, ataxia, muscle tremors, abnormal posture, and rigidity mainly in the hind legs. Some piglets are hyperexcitable; they may squeal and make paddling movements when they are handled. Approximately 1-10% of the piglets develop unilateral or bilateral corneal opacity, which usually regresses spontaneously. Other symptoms may include conjunctivitis, apparent blindness, nystagmus, constipation, and diarrhea. Affected piglets often die. The first piglets usually die within 48 hours of the onset of clinical signs; later, deaths are seen after 4 to 6 days of illness.

Weaned pigs more than 30 days old usually have transient, moderate symptoms that may include anorexia, fever, coughing, sneezing, and occasional corneal opacity. Neurologic signs are rare in this age group, but occasional depression, ataxia, circling or swaying of the head may be seen. On some poorly managed farms, a syndrome consisting of severe neurologic signs with a 20% mortality rate has been reported in 15-45 kg fattening pigs. On these farms, as many as 30% of the pigs may also develop corneal opacity.

Non-fatal reproductive failure is seen in older pigs. The symptoms include decreased conception rates, abortions, increased stillbirths and mummified fetuses in sows, and epididymitis, orchitis, and reduced semen quality in boars. Some animals may also have corneal opacity or mild anorexia.

Post Mortem Lesions
The typical lesions in suckling pigs are interstitial pneumonia and non-suppurative encephalomyelitis. Gross lesions may include signs of mild pneumonia (particularly at the ventral tips of the cranial lung lobes), congestion in the brain, and conjunctivitis and chemosis in the eye. The stomach may be mildly distended with milk, and the urinary bladder with urine. The peritoneal cavity sometimes contains a small amount of fluid with fibrin. The histopathologic lesions includes non-suppurative
encephalomyelitis; the gray matter of the thalamus, midbrain, and cerebral cortex is most often affected. The lungs may contain scattered areas of interstitial pneumonia, with thickened septae and a mononuclear cell infiltrate. Mild tonsillitis has also been reported.

The main necropsy lesion in experimentally infected boars is severe epididymo-orchitis. The testes may be atrophied. Histopathologic changes in the epididymitis may include spermatic granulomas and vacuolar degeneration of the ductular epithelium, associated with mononuclear cell infiltrates and interstitial fibroplasia. In the testes, degeneration of the seminiferous tubules and interstitial mononuclear cell infiltrates may be seen.

Lesions reported in experimentally infected gilts include focal congestion and hemorrhages in the placenta and endometrium. The fetuses may be dehydrated or mummified, or smaller than normal with dermal ecchymoses. Abnormal fetuses are interspersed randomly with normal fetuses.

Corneal opacity, characterized by anterior uveitis and corneal edema, may be present in pigs of any age.

**Morbidity and Mortality**

Outbreaks of blue eye disease can be seen throughout the year, but they are most common from April to July. Most outbreaks seem to be self-limiting. The mortality rate usually rises and falls within 2 to 9 weeks. Once the epidemic has ended, no more cases occur unless susceptible pigs are introduced to the farm.

In commercial breeding operations, the disease is usually noticed first in the farrowing unit, where large numbers of young piglets may die from encephalitis. Typically, 20-60% of the litters are affected. The overall morbidity rate in piglets is 20% to 50% and the mortality rate is approximately 90%; however, the severity of the symptoms varies with the age of the animals. Although severe disease can occur in piglets as old as 21 days, animals younger than 15 days are most susceptible. In one experiment, 3-day old piglets were all dead or dying within a week of inoculation, but only 30% of 17-day old piglets became ill.

In older animals, the virus seems to be cleared by the immune system. On most farms, the morbidity rate in weaned young pigs (more than 30 days old) is approximately 1% to 4%. The mortality rate in this group is usually low. However, on some poorly managed farms, severe neurologic signs with a 20% mortality rate have been reported in 15-45 kg fattening pigs. In adults, the only symptoms are non-fatal reproductive signs and occasional corneal opacity. A decrease in the conception rate usually persists for 6 to 8 months.

Persistent infections may be possible. Viral RNA has been found in pig tissues up to a year after infection, but it is not known whether the virus multiplies or is excreted.

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**Porcine Rubulavirus Infection**

**Diagnosis**

**Clinical**

Porcine rubulavirus infection should be suspected in an outbreak characterized by neurologic and respiratory disease in young piglets, reproductive failure in adult pigs, and corneal opacity in all ages.

**Differential diagnosis**

The differential diagnosis includes hemagglutinating encephalomyelitis virus infection and pseudorabies.

**Laboratory tests**

Serologic tests include hemagglutination inhibition, virus neutralization, indirect immunofluorescence, and enzyme-linked immunosorbent (ELISA) assays. All of the serologic tests detect seroconversion by the 8th day after infection.

The porcine rubulavirus can be isolated in pig kidney cell line (PK-15) cultures or chick embryos. Other pig cell lines and primary cultures, as well as baby hamster kidney cells (BHK 21) and Vero cell lines, are also susceptible. A rapid diagnostic test, which uses immunostaining to detect viral antigens in impression smears, is also available.

**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 should be collected for serology. In piglets, the porcine rubulavirus can be recovered consistently from the brain and tonsil, and sometimes from the lung, blood, spleen, liver, kidney, retropharyngeal lymph nodes, and nasal turbinates. It has also been found in various tissues of experimentally infected gilts including the lung, tonsils, ovary, placenta, uterus, and lymph nodes. The rapid immunostaining test uses lung, midbrain, or olfactory bulb tissue samples.

**Recommended actions if porcine rubulavirus infection is suspected**

**Notification of authorities**

Porcine rubulavirus infection must be reported immediately to state or federal authorities 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

**Quarantine and disinfection**

The porcine rubulavirus is contagious, and quarantine is necessary. Its disinfectant susceptibility has not been published; however, the related Newcastle disease virus,
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which also belongs to the genus *Rubulavirus*, is inactivated by formalin, phenol, or acid pH.

**Public Health**

Human infections have not been reported.

**Internet Resources**

World Organization for Animal Health (OIE)

http://www.oie.int

**References**


* Link defunct as of 2012