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Circoviridae

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This is a newly established family of very small, non-enveloped DNA viruses that contains three virus species of veterinary significance.

Viral Characteristics

- Very small (17 - 22 nm in diameter), naked icosahedral viruses with a circular, single-stranded DNA genome. The genome encodes a single capsid protein. See illustration of capsid, Fig. 8-1.
- Replication takes place in the nucleus of dividing cells and is similar to the parvoviruses.
- The single-stranded circular DNA of circoviruses is thought to be replicated by a rolling circle mechanism.
- In the cell nucleus, the ssDNA (either negative sense or ambisense) is used as a template for the formation of dsDNA by host repair enzymes. The dsDNA is then used as a template for both mRNA production (for translation of proteins) and copies of the genome for progeny virions. These products are self-assembled into complete progeny virions.
- Circoviruses are very stable in the environment; resistant to some disinfectants, including detergents.

Figure 8-1. Illustration of the capsid of a circovirus (17 - 22 nm). - To view this image in full size go to the IVIS website at www.ivis.org . -

Classification

The family has two genera based on genetic studies. *Gyrovirus* also differs from *Circovirus* in the replication cycle and the virions being larger. They are with their species as follows:

Circovirus
porcine *circovirus* type 1
porcine *circovirus* type 2
beak and feather disease virus
Gyrovirus
chicken anemia virus

Circovirus

As mentioned above, there are two porcine circoviruses:

- Porcine *circovirus* 1 (PCV 1) occurs widely in swine in Europe and North America; it produces non-clinical

infections.

- Porcine *circovirus* 2 (PCV 2) is the cause of postweaning multisystemic wasting disease (PMWS), which is discussed below.
  There is about 80% nucleotide homology between PCV 1 and PCV 2.
  Porcine *circovirus* 2 is antigenically distinct from PCV 1.

Porcine Multisystemic Wasting Syndrome (PMWS)

Cause

Porcine *circovirus* 2 (PCV 2). The current view is that porcine circovirus 2 (PCV 2) is necessary but not sufficient in itself to cause PMWS. As mentioned below other factors contribute to clinical disease.

Occurrence

Porcine multisystemic wasting syndrome is a worldwide, frequently occurring disease of pigs about six weeks of age. The disease occurs as a result of various stresses or concurrent infection with other agents, including porcine parvovirus or porcine reproductive and respiratory syndrome virus.

Transmission

It spreads horizontally and transplacental infections occur that may lead to abortion, weak neonates and mummified fetuses.

Pathogenesis

After initial infection there is viremia with spread to several organ systems, where lesions are produced.

Clinical & Pathologic Features

Weight loss, poor body condition, rough coat, diarrhea, debility, jaundice, lymphadenopathy (both T and B lymphocytes) and dysnea are characteristic.
There is granulomatous inflammation in several organ systems, which may include lung, kidney, liver, lymph nodes, spleen, tonsil, thymus and Peyer's patches. Large multiple basophilic intracytoplasmic inclusion bodies are frequently seen in macrophages and multinucleated giant cells.
The mortality rate may reach 10% in outbreaks.

Diagnosis

- Clinical specimens: preferably a whole pig; lungs, liver and kidney.
- A tentative diagnosis is based on age susceptibility, clinical signs and lesions.
- Definitive diagnosis is accomplished by fluorescent antibody staining of the virus in infected tissues.
- Immunofluorescence assay and ELISA are used to detect antibodies although the presence of specific antibody alone is not diagnostic.
- Although the virus can be cultivated in cells, virus isolation is not usually feasible for diagnostic laboratories. Many swine cells used for virus isolation are contaminated with PCV, which may lead to misdiagnosis.

Prevention

- Aqueous sodium chlorite solution is effective for the disinfection of premises. The virus is resistant to detergents.
- Removal of affected animals.
- An inactivated circovirus type 2 vaccine is available to aid in the prevention of PMWS. It is administered to piglets four weeks of age and older.

Psittacine Beak and Feather Disease

Cause

Beak and feather disease virus (Circoviridae).

Occurrence

Psittacine beak and feather disease (PBFD) affects many species of psittacine birds worldwide. Cockatoos are particularly susceptible.

Transmission

Virus spread is by direct and indirect contact. Infection occurs mainly by the alimentary and respiratory routes.

Clinical & Pathologic Features

The disease is seen in both chronic and acute fatal forms. The virus specifically infects the cells of the immune system and those cells that produce feathers and beak.
The cardinal signs of the chronic disease involve the beak and feathers. The beak deformities are characterized by palatine necrosis and elongated and easily fractured beaks. Abnormal feathering is progressive, becoming more evident with each

molt, and usually occurs in a symmetrical fashion with normal feathers being replaced by dystrophic feathers that cease to grow shortly after emerging from the follicle. The immune system is suppressed and secondary bacterial infections are common.
An acute form of PBFD occurs in which beak and feather lesions may not be evident. This form is seen most often in young birds and is characterized clinically by lethargy, anorexia, and diarrhea. Affected birds often die.

Diagnosis
- Clinical specimens: whole birds and infected feathers.
- Diagnosis is usually based on clinical signs and histopathologic examination of affected tissues.
- Intranuclear and intracytoplasmic inclusion bodies are commonly found in feathers, beak, and Bursa of Fabricius.
- Commercial tests utilizing PCR detect PBFD viral nucleic acid in diseased and asymptomatic birds. This is the best method available for detecting the presence of PBFD virus in the bird's blood when beak or feather lesions are not apparent.
- The virus has not been successfully propagated in embryonated eggs or cell cultures.

Prevention
- No vaccines are currently available.
- Prevention is best accomplished by good management practices. Note that virus particles can remain viable in the environment for months, long after the infected bird is gone.
- New additions to aviaries should only be purchased from reliable sources.
- It is advisable to quarantine and test birds for PBFD virus before introducing them to other psittacine birds.

Gyrovirus
Chicken Anemia Virus Infection
Cause
Chicken anemia virus (*Gyrovirus*).

Occurrence
A worldwide, common infection of chickens, particularly of commercial flocks and broilers. The virus may infect chickens of all ages.
Infections are most serious when there is concurrent infection with the infectious bursal disease virus, avian adenovirus, or reticuloendotheliosis virus.

Transmission
Direct and indirect spread by the oral-fecal and respiratory routes; also, vertically via the egg and via the semen of infected roosters. Laying hens thus infected are viremic for a period of 1 - 3 weeks. Chicks hatched from infected eggs are viremic and thus a source of infection.

Pathogenesis
The viremia developing in infected day-old chicks leads to infection of many organs and specifically T cells in the thymic cortex and bursa, and hemocytoblasts in the bone marrow.

Clinical & Pathologic Features
Overt disease is seen only in young chicks within the first 2 - 3 weeks of life. The virus is present in many organs and feces.
There follows immunosuppression and aplastic anemia with atrophy of lymphoid tissue.
Clinical signs begin at about two weeks of age and include anemia (pale), diarrhea, anorexia, depression and weight loss.
The mortality rate is usually about 10% but may be as high as 50% if there is dual infection.
Maternal antibodies prevent the development of clinical disease in chicks.
Necropsy lesions often noted are subcutaneous and muscle hemorrhages, pale visceral organs, an abnormal fatty-appearing bone marrow and thymic atrophy. Consistent microscopic lesions are found in the bone marrow where erythrocytes and other cells are replaced by fat cells and in the thymus, which is depleted of lymphocytes.

Diagnosis
- Clinical specimens: whole chicken, serum.
- Clinical signs, lesions and the aplastic anemia suggest chicken anemia virus infection.
- The virus can be cultivated in cells but isolation is not usually diagnostically feasible.
- Definitive diagnosis depends on the detection of viral DNA in the thymus or bursa by PCR, dot-blot hybridization or *in situ* hybridization.
- Serum antibodies can be detected by conventional procedures, such as ELISA.
- ELISA kits are available and are used to identify and eliminate positive hens before laying.

Prevention

- It is difficult to maintain laying flocks free of infection.
- A procedure used, is to deliberately expose layers before laying begins to infected tissue homogenates or litter from positive flocks.
- Losses are lessened if flocks are kept free of other immunosuppressive viruses.
- Antibiotics may be used to control secondary bacterial infections.
- Live vaccines are administered by injection or in drinking water to antibody-negative breeder flocks prior to the start of egg production.

Glossary

Aplastic anemia: An anemia in which the bone marrow fails to produce sufficient numbers of blood elements.
Dot-blot hybridization: A diagnostic procedure in which the material to be examined is blotted directly on to a membrane (frequently nitrocellulose) then hybridized with reference probes prepared from virus-specific DNA. The probes are labeled (chemically or radioactively) and a signal is detected where hybridization occurs.
Dystrophic: Maldevelopment caused by or related to faulty nutrition.