Avian Reo-virus infections

Dr./ Wafaa Abd El-ghany
Assistant Professor of poultry dis.,
Economic losses

- Increased mortality.
- Diminished weight gains, poor feed conversions.
- Uneven growth rates.
- Reduced marketability of affected birds due to downgrading at slaughter.
- Decreased egg production.
- Suboptimal hatchability/fertility, and vertical transmission of virus to progeny.
- Increased costs for poultry producers.
• Avian reoviruses are members of the Orthoreovirus genus in the family Reoviridae.
• These include a double stranded RNA (dsRNA) genome consisting of 10 segments packaged into a non-enveloped icosahedral double-capsid shell.
• The name "reovirus" derives from respiratory, enteric orphan, since they were first isolated from these sites in humans with initially, no apparent association with disease.
Cause

• Although they are considered to be ubiquitous in commercial poultry and for the most part appear harmless, avian reoviruses have been isolated from a variety of tissues and organs in chickens affected by assorted disease conditions, including viral arthritis/tenosynovitis, stunting, malabsorption syndrome, respiratory disease, enteric disease, immunosuppression, Hydropericardium and inclusion body hepatitis.
Susceptibility

• Chickens, turkeys, ducks and geese and he infection has been detected in several wild species of birds.
• Chicks are most susceptible to reovirus infection when they are very young and the development of joint lesions is a slow process.
• When viral arthritis does result from naturally occurring infection, it is usually not seen in young birds before 4-7 weeks old but may be seen in much older chickens as well.
Laboratory host system

• Reoviruses grow readily in the embryonating chicken egg following inoculation via yolk sac or chorioallantoic membrane (CAM).

• The yolk sac is preferred for original isolation and generally results in embryo mortality 3-5 days after inoculation, with affected embryos exhibiting a purplish discoloration due to massive subcutaneous hemorrhage.

• Mortality in CAM-inoculated embryos usually occurs on day 7-8 PI; embryos are slightly dwarfed with occasional enlargement of the liver and spleen. Necrotic foci may occur in both the liver and spleen, particularly in embryos that survive longer than 7 days PI. Small, discrete, slightly raised white lesions may be found on the CAM.
Laboratory host system

- The virus grows in primary chicken cell cultures of embryo, lung, kidney, liver, macrophages, and testicle.
- Primary chicken kidney cells from 2-6-week-old chickens are satisfactory, but for plaques and isolation, primary embryo liver cells are preferred.
- Chicken embryo fibroblasts are suitable for reovirus growth, but the virus often requires adaptation.
- Chicken-origin cell cultures infected with reoviruses are characterized by the formation of syncytia, which may occur as early as 24-48 hours, followed by degeneration, leaving holes in the monolayer and giant cells floating in the medium.
- Infected cells exhibit intracytoplasmic inclusions.
Mode of infection and transmission

- Horizontal transmission.
- Although reovirus may be excreted from both the intestinal and respiratory tracts for at least 10 days post-inoculation, virus generally appears to be shed from the intestine for longer periods, suggesting fecal contamination as a primary source of contact infection.
- Vertically transmission (the rate of egg transmission is low).
- The possibility of avian reoviruses entering through broken skin in the foot and localizing in the hock joint was demonstrated.
- Carrier birds as potential sources of infection as mechanical route of transmission due to the virus is resistant to inactivation.
Pathogenesis

- Viral infection through oral route inducing viraemia and localization of the virus in different tissues.
- The virus in the intestine causing enteritis.
- The virus in the pancreas and liver causes fibrosis and necrosis of pancreas.
- Pancreas secretes enzymes responsible for digestion, so obstruction of pancreatic ducts leads to mal-digestion and mal-absorption.
- Due to mal-digestion and mal-absorption stunting and abnormal feathering occur.
Viral arthritis
(Tenosynovitis)
Symptoms

1. Lameness and some birds show stunting.
2. Swelling in the area of the gastrocnemius tendon above the hock joint and the digital flexor tendon of the shank region and foot pad.
3. Rupture of gastrocnemius tendon:
   A) If unilateral, the affected leg can’t be extended and the bird can’t bear weight on this leg.
   B) If bilateral, the bird become immobilized.
4. Swelling and greenish discoloration of the skin over the site of the tendon rupture due to rupture of the blood vessels.
5. Morbidity can be as high as 100%, and mortality is generally less than 6%.
Lesions

1. Swelling and inflammation of the tendons and tendon sheath (above the hock joint along the posterior aspect of the shank).
2. Inflammatory exudates in the hock joints and tendon sheath.
3. Erosion and necrosis of articular cartilage of the hock joint.
4. Haemorrhages in synovial membrane above the hock joint and tendons.
5. Femur head necrosis.
Diagnosis

- Sampling from the joints, tendon sheath, synovial fluid.
- Isolation in ECE or TC (See Lab host system).
- Virus detection through AGPT, electron microscope, immunofluorescence or by (RT-PCR).
- Reovirus group-specific antibodies can be detected readily with the agar gel precipitin test, Virus neutralization, indirect fluorescent antibody (IFA) assay or ELISA test.
1. **Biosecurity measures:**
   - Following the removal of an infected flock from the premises, thorough cleaning and disinfection of a poultry house can prevent infection with pathogenic virus in subsequent groups.
   - Because of the relative stability of the avian reovirus group, commercially available disinfectants should be validated for efficacy before use. Lye and 0.5% organic iodine solutions are considered to be effective inactivating agents.
2. **Vaccination:**

- Vaccines and vaccination programs have evolved that are directed at providing protection at 1 day of age.

- Active immunization can be achieved by vaccination with viable attenuated (S1133)-derived reovirus vaccines that is usually applied by the subcutaneous route, although immunization by coarse-spray application of vaccine has also been used.

- Reovirus vaccination of breeding stock can be done with viable or inactivated vaccines or combinations of both.
2. **Vaccination:**

- The inactivated vaccines are more efficacious if preceded by vaccination with live vaccine.
- If a live vaccine is used, it should be administered prior to the onset of egg production to prevent transovarian transmission of the vaccine virus.
- The advantage of this program of vaccination is immediate protection of 1-day-old progeny provided by maternal antibody and a limitation of the potential for vertical transmission.
Mal digestion & Mal absorption syndrome
Stunting and runting syndrome
Helicopter disease
Pale bird disease
Brittle bone disease
Femur head necrosis
Infectious proventriculitis
Definition

• A disease syndrome caused by Reo-virus and characterized by uneven growth between the flock and poor feathering.
• This syndrome induced not only by Reo-virus alone but with other complicating viruses or bacteria (E. coli).
**Symptoms**

1. Un even growth (Runting and stunting).
2. Poor pigmentation especially of the shank (pale bird disease).
3. Abnormal feathering and run in different directions (Helicopter feathering).
4. Skeletal abnormalities as weak bone (Femur head necrosis).
5. Undigested food in the droppings (Mal-absorption syndrome).
Symptoms

1. Runting means decrease in body weight by 25-35%, while stunting means decrease in body weight by 50%.

2. Reo-virus interfere with the absorption of carotene, so the bird become pale.

3. Reo-virus interfere with the absorption of Ca, P and Vit. D, so rickets like symptoms appear (femur head necrosis) (weak bone).

4. Reo-virus interfere with the absorption of Sulpher containing amino acids that is important for feathers growth, so the bird show running of the feathers in different directions (helicopter feathering).
Lesions

- The pancreas is atrophied and fibrosed leading to shrinkage of the duodenum.
- Proventriculitis (enlarged proventriculus).
- Atrophy of the bursa and thymus glands.
- Necrosis, degeneration and fibrosis of the liver.
- The intestine show catarrhal enteritis, orange colour contents, undigested and poorly digested food especially at the rectum.
Normal (left) and affected (right) poventiculus
THANK YOU