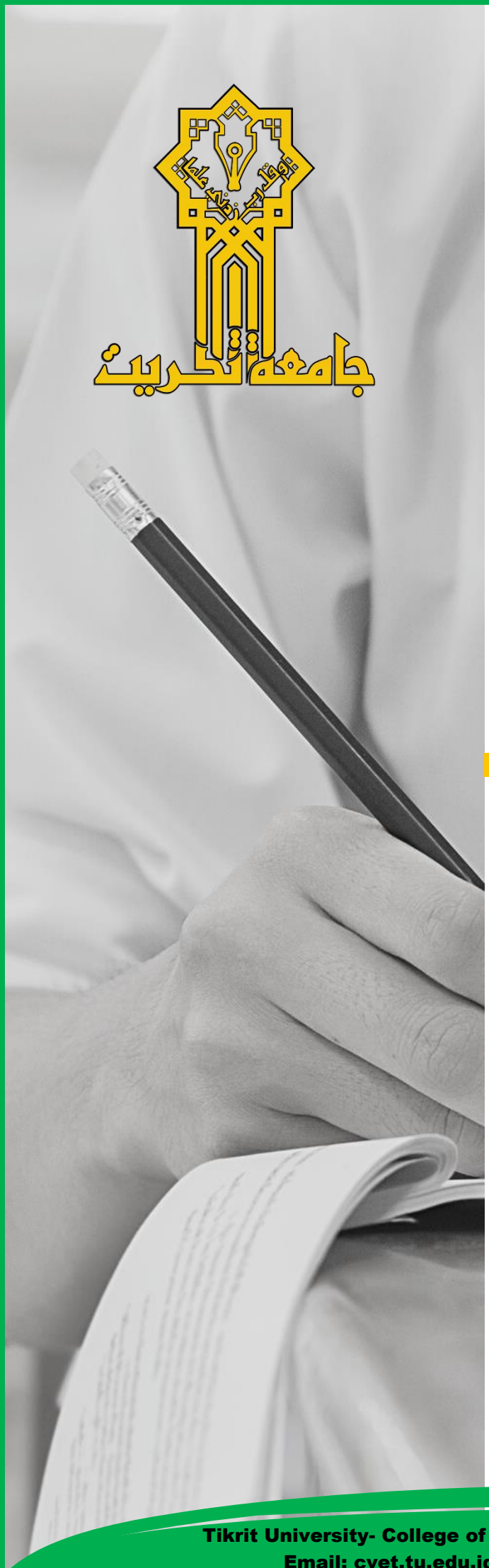

Tikrit University
College of Veterinary Medicine

Avian Reovirus Infections

Subject name: Poultry Diseases

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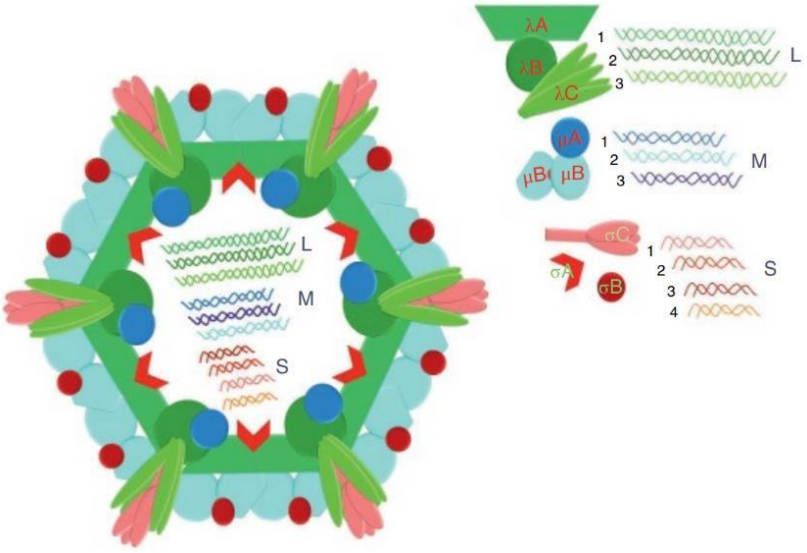
Avian Reovirus Infections

Summary

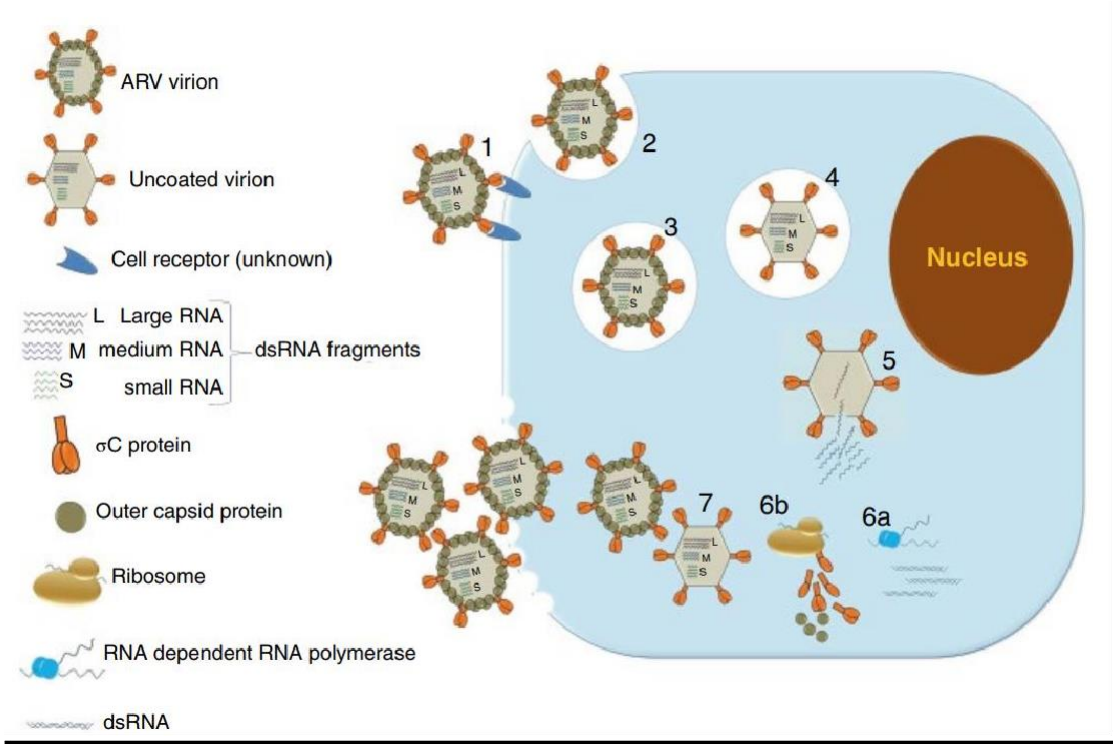
Avian reoviruses (ARVs) belong to the Reoviridae family in the genus Orthoreovirus. They are nonenveloped viruses composed of a double concentric icosahedral capsid with an external diameter of 80–85 nm that encloses 10 double-stranded RNA genome segments. The name "reovirus" is an acronym for "**r**espiratory, **e**nteric **o**rphan," The virus have possible cross-species infection is suggested. The ARV replicates in the gut of avian species and pathogenic strains can affect tendon and liver. The dsRNA segments are divided into three size classes: L1–L3 (large) encoding λa, λb, and λC structural proteins, M1–M3 (medium) encoding μA and μB structural proteins, and S1–S4 (small) encoding σA, σB ,and σC structural proteins. At least 12 primary translation products are expressed, of which 10 structural proteins.



Public Health Significance: There are no reports of ARV as being zoonotic agents.

Replication

The virus attaches to cellular receptors via the σC viral protein followed by receptor-mediated endocytosis. Following penetration into the host cell, the virus enters intracellular vacuoles where uncoating takes place. Following uncoating, viral cores are released into the cytoplasm. All ten viral mRNA are transcribed using the negative strand as template, catalyzed by virus-encoded dsRNA-dependent RNA polymerase. All transcripts are monocistronic are produced in similar amounts. This is followed by assembly of the virion within cytoplasmic inclusions the cores are assembled and then coated by outer-capsid polypeptides to generate mature virions



Susceptibility to Chemical and Physical Agents

Stable between pH3.0 and pH9.0 but are inactivated at 56°C in less than one hour. Avian reoviruses are relatively resistant to disinfectants. Reoviruses survived for at least 10 days on feathers, wood shavings, egg shells, and feed. In drinking water the virus was detectable for at least 10 weeks with little loss of infectivity.

Pathogenesis

Upon infection of the gastrointestinal tract, some ARV strains invade multiple organs of chickens and establish persistent infections. Reovirus isolated from intestinal contents of broiler chickens with malabsorption syndrome produced a transient. Avian reoviruses can be transmitted vertically via the egg as well as horizontally following the ingestion of infected fecal material. Inoculation of oral, subcutaneous, footpad, and intra-articular routes, produced arthritis/tenosynovitis with synovial hyperplasia and lymphocytic infiltration in all birds. One-day-old broiler chicks from vaccinated or unvaccinated breeder flocks were challenged with a virulent form of the vaccine strain. Chicks derived from the vaccinated flock, which had maternal antibodies, had reduced tenosynovitis lesions by about 50%. The rate of virus recovery from the hock joints, however, was similar in both groups. The virus is able to infect older birds but the disease generally is less severe and the incubation period is longer. The pathogenicity enhanced by coinfection with Eimeria spp., infectious bursal disease virus, chicken anemia virus, Escherichia coli, and common respiratory viruses. Cryptosporidium baileyi produced systemic infection

Incubation Period:

The incubation period differs depending upon the virus pathotype, age of host, and route of exposure. For 2-week-old chickens, the incubation period varied from 1 day (footpad inoculation) to 11 days (intramuscular, intravenous, intrasinus inoculation). Mature birds inoculated by oral and respiratory routes showed the presence of virus at 4 days PI. The rate of virus isolation was greatly reduced by 2 weeks, and no virus was present at 20 days PI.

Clinical Signs:

1- Morbidity can be as high as 100% while mortality is generally less than 6%.

2- lameness usually appears at 4–5 weeks of age although the infection may have occurred soon after hatching.

3- In acute infections, some chickens also may be stunted.

4- delayed growth, low uniformity, lethargy, and watery diarrhea

5- mild respiratory disease in baby chicks

6-

Gross (P.M.) lesions:

A-Acute stage:

1- The most severely affected birds exhibit swollen hock joints and enlargement in the area of the gastrocnemius or digital flexor tendons.

2- Rupture of the gastrocnemius tendon may occur in heavy birds.

3- In severe cases, birds are immobilized and may be recumbent close to drinkers.

4- The typical uneven gait in bilateral rupture of the tendon results from the inability of the bird to mobilize the metatarsus. It is often accompanied by ruptured blood vessels.

5- The affected joints usually feel warm.

6- The rupture of gastrocnemius tendon is often perceived as a greenish discoloration of the skin due to extravasation of blood.

7- Removal of the skin at necropsy will reveal the broken end of the tendon.

8-Gelatinous fluid or blood- tinged exudate over the joint portion.

9-Considerable amount of purulent exudate resembling that seen with mycoplasma synovitis.

10-Marked edema of the tarsal and metatarsal tendon sheaths.

11-Petechial hemorrhages are frequent in the synovial membranes above the hock.

12-Hepatitis, ascites, hydropericardium, pale kidneys, and depleted bursas.

13-Enlarged proventriculus, pancreatic atrophy, and bone abnormalities.

B-Chronic stage:

1-Hardening and fusion of tendon sheaths.

2-Small, pitted erosions develop in the articular cartilage of the distal tibiotarsus.

3-The erosions enlarge, coalesce, and extend into underlying bone.

4-overgrowth of fibrocartilaginous pannus develops on the articular surface.

5-The diaphysis of the proximal metatarsal of the affected limb is also enlarged.

6-Condyles and epicondyles are frequently involved.

Histologic changes:

A-Acute phase:

1-Edema, coagulation necrosis, heterophil accumulation, and perivascular infiltration are seen along with hypertrophy and hyperplasia of synovial cells, infiltration of lymphocytes and macrophages, and proliferation of reticular cells.

2-Parietal and visceral layers of the tendon sheaths to become markedly thickened.

3-The synovial cavity is filled with heterophils, macrophages, and sloughed synovial cells. Periostitis is characterized by increased osteoclasts activity.

4-Histopathologic changes include hepatitis, nephritis, myocarditis, pericarditis, catarrhal enteritis, pancreatic necrosis, and encephalomalacia.

B-Chronic phase:

1-The synovial membrane develops villous processes, and lymphoid nodules are seen.

2-Increase in fibrous connective tissue and a pronounced infiltration or proliferation of reticular cells, lymphocytes, macrophages, and plasma cells

3-Some tendons are replaced completely with irregular granulation tissue.

4-Chronic fibrosis of tendon sheaths, with fibrous tissues invading tendons and resulting in ankylosis and immobility.

5-Osteoblasts become active and lay down a thickened layer of bone beneath the erosion. Osteoblastic activity is present on the condyles, epicondyles, and accessory tibia, producing osteoneogenesis.

Diagnosis:

1-Clinical signs and gross and histologic lesions.

2-virus isolation

3- RT-PCR.

Differential Diagnosis

1- Mycoplasma
2- Newcastel Disease
3- Metablic diseases
4- IBD
5- 5-HHS.

Treatment

1- Antibiotics:
2- Anti-inflammatory:
3- Supplements:

Prevention and Control:

1- Eliminating reservoirs
2- Good management practices, with emphasis on sanitation
3- Biosecurity
4- Water fountains should be self-cleaning, and feeders should be covered to prevent contamination as much as possible.
5- Free-flying birds, rodents, and other animals can be excluded.
6- All housing and equipment should be cleaned and disinfected before repopulation.

Vaccination

-Vaccination of breeders to induce high levels of neutralizing antireovirus antibodies.

-Maternal antibody half-life in the chick is about 5 days, and at 10–15 days of age the level of maternal antibody is nonprotective

-The live attenuated vaccines that are in use worldwide are based on S1133 strain of ARV that was developed in 1983. Until recently, most inactivated vaccines were derivatives of S1133, such as strains 2177, 1733, and 2408.

-Live vaccines should be administered only prior to the onset of egg production in order to prevent trans-ovarian transmission of the vaccine virus.

-The common protocols for vaccination of broiler breeders include 1–3 live attenuated vaccines up to 12 weeks of age followed by 1–3 inactivated vaccines.

-Live and inactivated vaccines are administered via intramuscular or subcutaneous routes.

-In ovo vaccination.