Tikrit University
College of Veterinary Medicine

Adenovirus Infections Quail Bronchitis

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Lecturer name: Ismael I. Hasan

Academic

Email:ismailhasan@tu.edu.iq

Lecturers link

Adenovirus Infections
Quail Bronchitis

Summary

Quail bronchitis (QB) is a naturally occurring, acute, highly contagious, fatal respiratory disease of young bobwhite quail (*Colinus virginianus*). Quail bronchitis is characterized by rapid onset and high morbidity and mortality, and mainly affects captive-reared birds. The etiologic agent is quail bronchitis virus (QBV). Quail bronchitis virus and chicken embryo lethal orphan (CELO) virus, both aviadenoviruses, are considered to be the same agent. It contains a DNA genome and is icosahedral and nonenveloped, and ranges in size from 69–75nm in diameter.

Public Health Significance:

Non-significant

Replication General

adeno

Susceptibility to Chemical and Physical Agents

General adeno

Transmission:

Vertical (rare) & Horizontal

Incubation Period:

Following infection, signs may develop as early as 2 days but generally develop in 3–7 days. Severity of infection varies depending on the age at which the bird is infected. Quail bronchitis is most severe in quail less than three weeks of age.

Clinical Signs:

1- Manifested by respiratory distress that leads to death in young quail.

2- Sudden onset of rales, sneezing, or cough ing that spreads rapidly through the flock and results in mortality.

3- Excess mucus in the trachea, bronchi, and air sacs

4- Morbidity and mortality from field cases frequently exceed 50% and may be much higher in flocks affected at younger than 3 weeks of age.

5- Older birds frequently are asymptomatic but develop antibodies to group I/serotype 1 adenovirus

6- Decreased feed consumption, ruffled feathers, huddling under brooders, wing droop, open-mouthed breathing, "snicks," and nasal–ocular discharge.

Gross (P.M.) lesions and Histologic changes (Microscopic Lesions):

1- Nasal–ocular discharge also may be noted.

2- Opacity and filling of the trachea by pale, moist, necrotic, and some times hemorrhagic exudate is common.

3- On cross section, the mucosa is markedly thickened.

4- Similar exudate may be found in the anterior air sacs.

5- Histologically, tracheal lesions may include epithelial deciliation, cell swelling, karyomegaly, necrosis, desquamation, and leukocyte infiltration.

6- Basophilic, intranuclear viral inclusions are common in intact or desquamated tracheal epithelium.

7- Electron microscopic changes are similar to those seen histologically but also demonstrate phagocytosed viral particles.

8- In the lungs, red, consolidated areas surround the bronchial hilus.

9- On section, bronchi frequently contain exudate similar to that in the trachea, indicative of a necrotizing, proliferative bronchitis.

10- Inflammatory exudates consisting of lymphocytes, heterophils, and fluid may extend into the surrounding pulmonary parenchyma, but the intensity of the leukocyte response varies and may be confounded by secondary bacterial infections.

11- Histologically, bronchial changes are similar to those in the trachea, except that bronchi may demonstrate more epithelial proliferation. Most lesions are associated with large basophilic intranuclear inclusions.

12- Lesions in the liver include multifocal pale, pinpoint to 3mm necrotic foci. Histologically, these foci are characterized by hepatocellular necrosis, infiltrated to varying degrees by lymphocytes and fewer heterophils. Inclusion bodies are occasionally seen in hepatocytes adjacent to necrotic foci and/or biliary epithelium.

13- Lesions occur in the spleen and cloacal bursa but can be difficult to identify in quail less than three weeks of age. The spleen may be mottled and slightly enlarged. Histologically, affected spleens have multifocal, often extensive zones of necrosis, characterized by lymphocytolysis with

increased fibrillar eosinophilic intercellular material, with minimal leukocyte infiltration. Adenoviral inclusions are rare in the spleen.

14- Histologic lesions of the cloacal bursa include necrosis of lymphocytes, frequently accompanied by generalized lymphoid depletion and follicular atrophy. Intranuclear viral inclusions are common in bursal epithelium. Experimentally, some quail also develop necrotizing pancreatitis associated with adenoviral inclusions

Diagnosis:

1- Clinical signs and gross and histologic lesions.
2- Virus isolation
3- PCR.
4- Serology

Differential Diagnosis

1- Bacteremia (spleen).
2- Acute viral (highly pathogenic avian influenza, HPAI; Newcastle disease, ND).
3-Parasitic (coccidiosis).
4- Toxic (heavy metals, sulfa drugs).
5- Postmortem autolysis of the intestinal tract.
6- Respiratory diseases including AI, ND, Syngamus trachea.
7- Carbon monoxide.
8- Embryo mortality (IB,ND).
9- Pulmonary aspergillosis
10-ILT, AI

Treatment

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

Prevention and Control (Intervention Strategies):

1- Management Procedures

2- **Vaccination:**

Clinically non-significant because the duration of immunity in QB is not known, but survi vors of both naturally occurring and experimental infec tions were refractory to challenge with QBV for at least six months, and significant antibody levels developed in serum of quail following infection. Young chicks with maternal antibody also are refractory to challenge with QBV, but maternal antibody is not believed to prevent virus multiplication.