Infectious Bursal Disease (IBD, Gumboro)

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General Information

Common in all over the world

Very contagious

Clinical: depression and death

Subclinical:immunosuppression

Significant economic losses

Etiology

Birnaviridae (family)
- *Avibirnavirus (genus)*
- **Infectious bursal disease virus (IBDV)**

- Double-stranded RNA (dsRNA)
- Bi-segmented genome
  - Segment A
  - Segment B
- **Segment A:** Encodes a polyprotein that is cleaved into:
  - VP2 – Major capsid protein, highly immunogenic, contains neutralizing epitopes
  - VP3 – Internal structural protein, binds viral RNA
  - VP4 – Viral protease, responsible for cleavage of the polyprotein

  Also encodes:
  - VP5 – Non-structural protein, involved in virus release and virulence
- **Segment B:**
  - Encodes VP1, the RNA-dependent RNA polymerase (RdRp)
  - Essential for viral replication

Etiology

Viral proteins

- VP1: RNA polymerase
- VP2: Major antigenic protein, vaccine target
- VP3: RNA-binding structural protein
- VP4: Viral protease
- VP5: Virulence-associated, apoptosis-related

Etiology

- IBDV is antigenically divided into two serotypes:
  - serotype 1 and serotype 2.
  - Gumboro disease in chickens is caused exclusively by **serotype 1**
- Based on antigenicity and pathogenicity serotype 1 IBDV strains are classified as:
  - Classical IBDV
  - Variant IBDV
  - Attenuated IBDV
  - Very virulent IBDV (vvIBDV)
  - New variant IBDV
- The virus is highly stable and resistant to ether and chloroform
- It maintains stability over a wide pH range (pH 3–9)
- IBDV can remain viable in poultry houses for 3–4 months.

Epidemiology

- Transmitted mainly via the fecal–oral route through infected feces, direct contact, contaminated equipment, workers, feed, and water
- Less frequently enters via the conjunctiva and respiratory tract
- No vertical transmission has been reported
- Dogs and cats may act as mechanical carriers after consuming infected carcasses
- Morbidity can reach up to 100%
- Infects domestic and wild birds, but clinical Gumboro disease occurs **only in chickens**
- First reported in 1962 in Gumboro, England; spread across Europe in the 1970s
- Approximately 60% of global isolates are related to the vvIBDV genotype

Immun system and Immunsupression

Target organ: Bursa of Fabricius

- Around days 2–3 post-infection, due to edema and hyperemia, the bursa enlarges, shows gelatinous transudate, color changes from white to cream/yellow, and hemorrhages may occur
- Up to a 2-fold enlargement is observed on day 4
- Bursa size returns to near normal by day 5
- Marked atrophy occurs, reaching approximately one-third of the normal size by day 8
- Enlargement of the spleen and hemorrhages in the proventriculus and gizzard may be observed
- In vvIBDV infections, the thymus, caecal tonsils, spleen, and bone marrow are also adversely affected, leading to severe immunosuppression

Clinical Signs

- **In the acute form**, chickens show a sudden onset of depression, lethargy, ruffled feathers, and high fever.
- Watery white diarrhea contaminated around the cloaca due to excessive urate crystals is commonly observed.
- Polyuria and dehydration develop rapidly, and the disease may result in death, especially in young birds.
- **The subclinical form** is the most common presentation, particularly in young chickens infected early in life.
- Although clinical signs may be absent or mild in the subclinical form, the virus causes severe immunosuppression by destroying immature B lymphocytes in the bursa of Fabricius.
- As a result, affected birds become highly susceptible to secondary infections and show poor responses to vaccination.

Diagnosis

Clinic and necropsy

- Clinical form
- Subclinical form

Laboratory diagnosis

- Sample

Molecular analysis and typing

- RT-PCR and sequencing

Control Program

Breeding

Vaccination programs

Serological monitoring

Broiler

Vaccination programs

Maternal antibodies

Serological monitoring

Diagnosis and monitoring of the virus

RT-PCR

Typing

Monitoring of Immunosuppression

Clinic-necropsy

Histopathology

CHICKEN INFECTIOUS ANEMIA DISEASE

- Aplastic anemia in chicks
- Atrophy of lymphoid organs
  - Thymus
  - Bursa of Fabricius
  - Spleen
- Severe immunosuppression
- Etiologic agent
  - Initially identified as Chicken Anemia Agent (CAA)
  - Later named Chicken Anemia Virus (CAV)
  - Currently referred to as Chicken Infectious Anemia Virus (CIAV)
- Epidemiology
  - Reported worldwide
  - Also reported in Turkey

Etiology

- *Anelloviridae* (family)
  - *Chicken infectious anemia virus* (CIAV)
- Contains single stranded DNA
- Serologically uniform
- Marked differences in pathogenicity among strains
- Classified into four major genotypes:
  - Genotype I, Genotype II, Genotype III, Genotype IV

Etiology

- Three viral proteins: VP1, VP2, VP3

- VP1
  - Major capsid protein
  - Associated with virulence and viral replication

- VP2
  - Non-structural protein
  - Essential for protective immunity

- VP1 & VP2: Required for induction of neutralizing antibodies

- VP3 (Apoptin)
  - Induces apoptosis in chicken thymocytes and lymphoblastoid cells
  - Binds to cellular DNA

Epidemiology

- Common in the world
- Chickens of all ages are susceptible to infection
- The agent can be transmitted **vertically** and **horizontally**
  - Fecal–oral route via virus shed in feces of infected birds
  - Direct or indirect contact
  - Respiratory route
  - Via infected feather follicle epithelium
  - Through contaminated fomites
- The presence of the virus in rooster semen is important for transmission.

Clinical Findings

- Anorexia, lethargy, depression
- Ruffled feathers
- Pale comb, wattles, beak, and mucous membranes
- Hemorrhages in the skin, subcutaneous tissue, and muscles
- Increased mortality (Mortality rate: 10–60%)
- Growth retardation and paralysis
- **Anemia is the most prominent clinical finding**
- Anemia peak severity: 14–16 days post-infection

Necropsy Findings

- Moderate to severe atrophy is the most commonly observed lesion
- **Bone marrow atrophy is the most characteristic finding (e**specially evident in the femoral bone marrow)
- Bone marrow changes from red, gelatinous consistency to pale yellow or whitish fatty appearance
- Lesions may vary in the presence of secondary infections

Diagnosis

Clinical and necropsy

Laboratory examinations

- Virus isolation
- Serology
- Molecular analysis

Protection and Control

Vertical transmission must be considered

Vaccination

ADENOVIRUS INFECTIONS in CHICKENS (Fowl Adenovirus)

Adenovirus (FAdV) infections

- Hepatitis-Hydropericardium Syndrome (HHS)
- Hepatitis with Inclusion Bodies (HIB)
- Adenoviral Gizzard Erosion (AGE)
- Infectious Salpingitis (Egg Drop Sydrome, EDS)
- Turkey Hemoragic Enteritis, THE

Etiology

- *Adenoviridae* (family)
  - ***Fowl adenovirus, FAdV***
- Genetic material DNA
- Classified into 5 genotypes and 12 serotypes
- Composed of 13 structural proteins
- Highly resistant to desiccation in the environment
  - Resistant to phenol (2%) and alcohol (50%)
  - Sensitive to aldehyde and iodine
- Remain infective for up to 7 weeks in contaminated carcasses and feces

Etiology

Classification of Avian Adenoviruses

- Group I (Aviadenovirus)
  - Most common in poultry
  - Includes viruses causing: Inclusion Body Hepatitis (IBH), Hydropericardium Syndrome (HHS), Adenoviral Gizzard Erosion (AGE)
- Group II (Siadenovirus)
  - Includes viruses causing: Turkey Hemorrhagic Enteritis (THE), Marble Spleen Disease (MSD) in pheasants
- Group III (Atadenovirus)
  - Includes the virus causing: Egg Drop Syndrome 1976 (EDS-76)

Epidemiology

- Widely detected in different poultry species, including:
  - Chickens
  - Turkeys
  - Ducks
  - Geese
- Transmission routes
  - Vertical transmission
  - Horizontal transmission : Respiratory secretions, Fecal contact, Contaminated fomites
- Both vertical and horizontal transmission play an important role in:
  - Inclusion Body Hepatitis (IBH)
  - Hydropericardium Syndrome (HHS)

Aviadenovirus Infections

1. Inclusion Body Hepatitis (IBH)

- Reported worldwide in poultry
- Rarely observed in turkeys
- **Clinical signs:** Lethargy, Hunched posture, Ruffled feathers, Distended abdomen, Yellow, mucoid diarrhea
- **Necropsy findings**: Enlarged, pale, necrotic, friable, and hemorrhagic liver, Petechial and ecchymotic hemorrhages in skeletal muscles

Aviadenovirus Infections

2. Hepatitis-Hydropericardium Syndrome (HHS)

- Horizontal transmission is highly important
- Main causative agent: Fowl adenovirus group C, serotype 4 (FAdV-4)
- Occasionally reported in Ducks, Pigeons, Quails
- **Clinical signs:** Acute course with high morbidity and mortality, Ruffled feathers, Yellow, mucoid diarrhea, Depression, weakness, and sudden death
- **Necropsy findings:** Hydropericardium: straw-colored, watery or jelly-like fluid in the pericardial sac, Hemorrhages in the heart muscle and other organs, Pulmonary congestion and edema, Enlarged, pale, friable liver, Pale kidneys, Enlarged, mottled spleen, Swollen bursa of Fabricius

Aviadenovirus Infections

3. Gizzard Erosion (GE)

- No marked clinical signs
- Weight loss may be observed
- Disease is usually detected at slaughter
- **Necropsy findings**: Brown to black erosive lesions in the gizzard, Perforation, roughening, and discoloration of the koilin layer

Siadenovirus Infections

1. Turkey Hemorrhagic Enteritis (THE)

- Causative agent: Turkey adenovirus A
- Turkeys are the natural host
- Transmission: fecal–oral route
- **Clinical signs**: Severe bloody diarrhea, Feathers around the vent covered with blood
- High mortality (up to 60%)
- **Necropsy findings**: Lesions mainly in the spleen and intestines, Severe splenic congestion with grayish necrotic foci, Markedly distended intestines, filled with unclotted blood, Chronic enteritis

Siadenovirus Infections

2. Marble Spleen Diseases (MSD)

- Acute respiratory disease of pheasants
- Characterized by depression, enlarged, mottled (“marble”) spleen, pulmonary congestion and death
- Causative agent: *Turkey adenovirus 3 (TAdV-3)*
- **Clinical course:**
  - Dyspnea due to pulmonary edema,
  - Asphyxiation and sudden death

Atadenovirus Infections

Egg Drop Syndrome 76 (EDS-76)

- Causative agent: *Duck adenovirus 1*
- Differs from other avian adenoviruses by its strong hemagglutination of avian erythrocytes
- **Three disease models of EDS in chickens**

1. Classic EDS-76:

- Vertically transmitted
- Affects egg production
- Characterized by sudden drop in egg production, Thin-shelled, soft-shelled, or shell-less eggs

Egg Drop Syndrome 76 (EDS-76)

2. Endemic EDS-76:

- infection occurs as a result of horizontal transmission during the laying period.

3. Sporadic EDS-76:

- transmission occurs through direct contact with domestic ducks or geese, or via water sources contaminated with feces of wild birds

Clinical Signs

- In affected flocks, a transient loss of eggshell pigmentation is observed initially
- Followed by production of:
  - Thin-shelled eggs
  - Soft-shelled eggs
  - Shell-less eggs
- Transient diarrhea and depression may occur in chickens
- Decreased egg production
- Failure to reach peak egg production

Diagnosis

Clinical symptoms

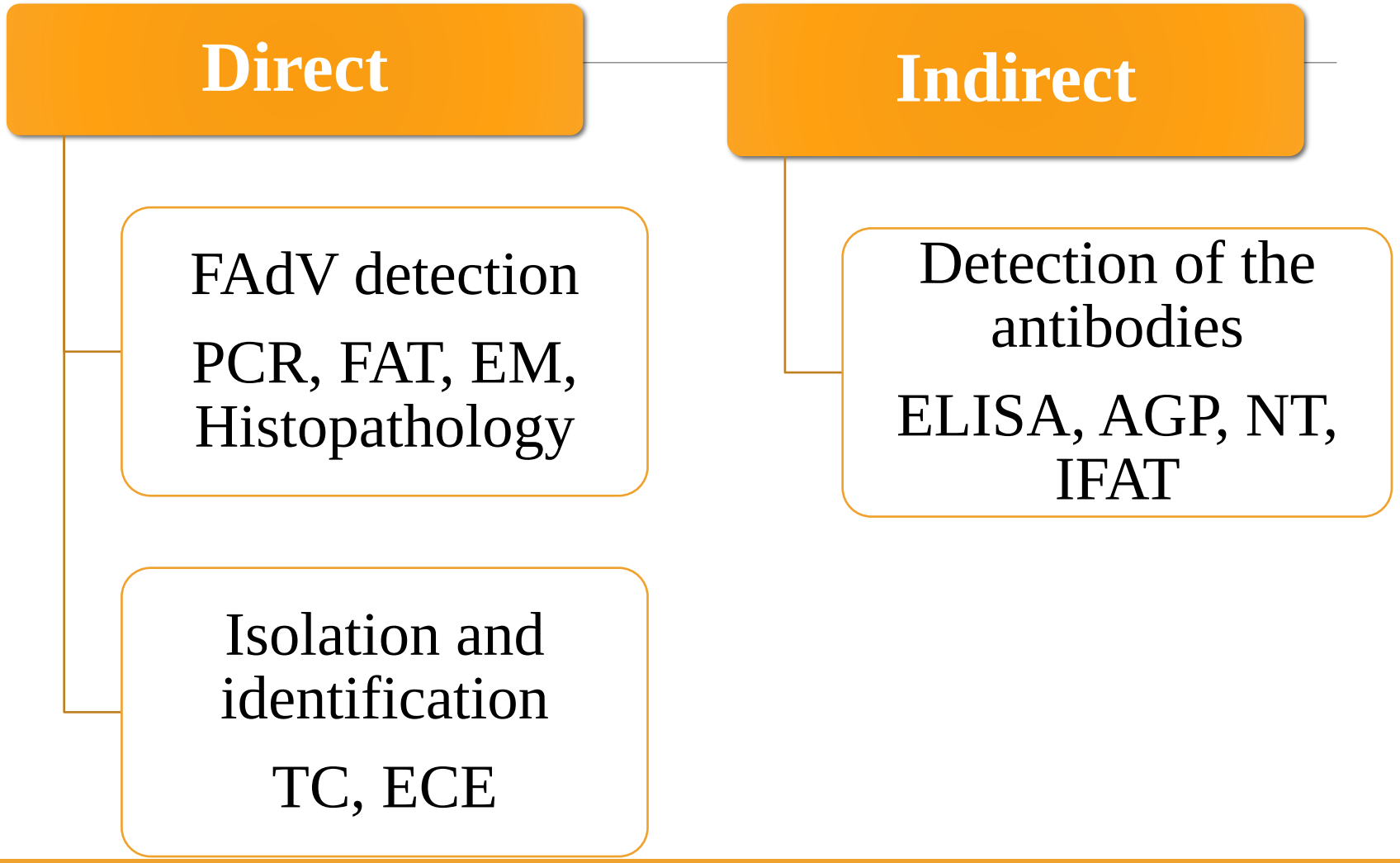
- Age
- Mortality

Necropsy (macroscopic findings)

Laboratory Examination

- Organ
- Serum

Laboratory Examination



Molecular Diagnosis and Typing

PCR

RFLP

Sequence analysis

Control

Transmission route

- Vertical
- Horizontal

Biosecurity

Vaccination

- Serotype(s)
- Inactivated vaccines
- Live vaccines
- Subunit vaccines

REOVIRUS INFECTIONS

Etiology

- *Reoviridae (*family)
  - *Sedoreovirinae* (subfamily)
  - *Spinareovirinae* (subfamily)
    - *Orthoreovirus (genus)*
      - *Avian reoviruses → Avian orthoreovirus*

- Nucleic acid with double-stranded RNA
- Poultry reoviruses are resistant to ether, chloroform, low pH such as pH 3, trypsin and sodium deoxycholate
- The virus is also highly resistant to heat inactivation
- Unlike mammalian reoviruses, avian reoviruses do not have hemagglutination and hemadsorption properties

Epidemiology

- Transmission is multifactorial
- Occurs via vertical and horizontal routes
- Horizontal transmission includes:
  - Fecal–oral route
  - Skin contact
  - Respiratory route
- ARV can survive for prolonged periods on:
  - Feathers
  - Eggshells
  - Drinking water
  - Poultry house equipment

Clinical Symptoms

Reovirus infections are seen in different diseases in chickens

- **Arthritis and tenosynovitis**
- **Runting–Stunting Syndrome (RSS)**
- **Hepatitis**
- **Respiratory tract infections**
- **Enteric infections**
- **Malabsorption syndrome**

Diagnosis

1. **Clinical and necropsy findings**
2. **Laboratory examinations**

a) Virus isolation

b) Molecular analysis

c) Serological tests

Protection

Vaccines

- Live
- Inactivated