Avian encephalomyelitis (AE)

Epidemic tremor

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Definition

• Avian encephalomyelitis (AE) is a viral infection affecting primarily young chickens and characterized by ataxia, especially of the head and neck and drop in egg production in layers.
Cause

- Enterovirus of the Family Picornaviridae.
- The virus could be propagated in the yolk sac of ECE after adaptation inducing leg paralysis and muscular dystrophy of the embryos 10-12 days post-inoculation.
- If there is no lesions in the embryo, hatched chicks will show the characteristic clinical signs.
- Fibroblasts, kidney cells, and neuroglial cells from chicken embryos and pancreatic cells from young chicks were used to cultivate both adapted and field strains of virus.
Susceptibility

- Primarily young chickens (1-6) weeks old birds.
- Pheasants, quails and turkeys could take natural infection also but less susceptible than chickens.
- Duckling, pigeons and G. fowl can be experimentally infected.
- Laboratory animals are refractory to intracerebral inoculation.
Mode of infection and transmission

- Vertical transmission from infected hens to embryos and consequently chicks is the most common route of infection.
- Infection in incubators at hatching or in the brooder by contact with infected chicks.
- Transmission through direct contact.
- Ingestion of contaminated food and water by the droppings of infected birds.
- Infected birds harbor the virus in intestine and shed it in the faeces for 10-15 days, as the virus can survive for at least 3 weeks in droppings.
Incubation period

- Under vertical transmission, the incubation period is 1-7 days.
- Contact or oral transmission, the incubation period is 11 days.
Diagnosis

• **History:**
  1. Nervous manifestation of young chickens (1-6 weeks old).
  2. Laying hens show transient drop in egg production without signs, lowering in hatchability and hatched chicks from these eggs show ataxia and leg paralysis.
Diagnosis

- **Symptoms:**
  1. Young chickens (up to 6 weeks old):
     - General symptoms.
     - Nervous manifestations:
       A) Intermittent and rapid tremors of the head and neck.
       B) Progressive ataxia.
       C) Incardination in movement.
       D) Paresis or paralysis of legs.
       E) Prostration (set on hock joint) and death.
     - Mortality rate 5-10% with maximum of 50-60%.
     - Morbidity rate 40-60%.
     - Some recovered birds show blindness due to opacity of the eye pupil (cataract) of one or both eyes.
Diagnosis

• **Symptoms:**

2. **After 6 weeks old (adult laying hens):**
   - Transient drop in egg production up to 5-10% for 10-15 days.
   - Lowering in hatchability due to embryonic deaths during last 3 days of incubation (lae embryonic death).
   - Chicks hatched from these eggs develop ataxia and leg paralysis.
Iridocyclitis and cataracts and general eye enlargement.
Locally extensive cataract

Cataract formation in the affected eye on the left,

focal cataract
Diagnosis

• **Post-mortem lesions:**
  - No post-mortem lesions in young’s or adult but sometimes necrosis of the gizzard muscles (whitish areas) (masses of lymphocytic infiltration) could be seen.

• **Histopathological examination:**
  - The brain and spinal cord of the affected chicks in H&E stained sections microscopically show Gliosis, pre-vascular cuffing and neural degeneration.
  - Lymphocytic infiltration in the proventriculus, gizzard and pancreas could be seen.
Ventriculus (gizzard): Moderate locally extensive ventriculitis
The cerebellum of the abnormal embryo has herniated out of the Foramen Magnum and is extensively swollen, glistening, and wet.

The brain of the chick on the right has lost architectural detail, is soft, and pale yellow.

Extensive malacia
Brain: within normal limits

Brain: Focal lymphoplasmacytic perivascular cuff
Brain, white matter: Glial nodule and diffuse gliosis
Moderate subacute multifocal lymphocytic ventricular myositis

Muscle: Within Normal Limits
Diagnosis

• The brain is an excellent source of virus for isolation, although other tissues and organs, the pancreas and duodenum were especially reliable sources of virus.

• To inoculate embryos (obtained from a susceptible flock) via the yolk sac when 5-7 days of age, allow these to hatch, and observe chicks for signs of disease during the first 10 days. When clinical signs appear, brain, proventriculus, and pancreas should be examined for lesions."

• Brain, pancreas, and duodenum from affected chicks can be examined for specific viral antigen by immunofluorescence, immunofluorescence, and ELISA techniques.
Stunting with limb malformation (muscular dystrophy)
Diagnosis

- To determine immunity of a flock is the embryo or egg susceptibility (ES) test.
- Fertile eggs from the flock to be tested are incubated, along with control eggs from a known susceptible flock. After six days, each embryo is inoculated via the yolk sac with 100 EID50 of egg-adapted virus. Embryos are examined 10-12 days PI for characteristic lesions.
- If 100% of embryos are affected, the flock is considered susceptible; less than 50% affected indicates immunity. Intermediate figures should be considered non-definitive and may indicate recent exposure.
Differential diagnosis

1. **Nutritional diseases:**
   A) **Vit. E def. (Encephalomalacia):**
   - Therapeutic diagnosis.
   - Negative virus isolation.
   - Microscopic exam. (degenerative changes).
   B) **Vit. B2 (riboflavin) def:**
   - Therapeutic diagnosis.
   - Negative virus isolation.
   - Microscopic exam. (degenerative changes).
   - Bilateral affection of the sciatic nerves.

2. **Viruses affections:**
   A) **MD:**
   B) **ND:**
   C) **AI:**
Control

• No satisfactory treatment is known for acute outbreaks in young chicks.

• Removal and segregation of affected chicks may be indicated under certain conditions, but they generally will not develop into profitable stock.

• After a flock has experienced an outbreak of AE, no further evidence of it is likely to be observed.
Prevention

- Control of AE is achieved by vaccination of breeder flocks during the growing period to ensure that they do not become infected after maturity.
- Vaccination preventing dissemination of the virus by the egg-borne route.
- Also, maternal antibodies protect progeny against contact to AEV during the critical first 2-3 weeks.
- Vaccination may also be used with commercial egg laying flocks to prevent a temporary drop in egg production associated with AE.
Prevention

• Inactivated vaccines have been developed and may be useful in flocks already in production or where the use of a live virus is contraindicated.

• Most flocks, however, are vaccinated with a live, embryo-propagated virus, such as strain 1143, which can be administered by naturally occurring routes such as via drinking water or by spraying.

• Live virus vaccines, which can be stored frozen or after lyophilization are similar to field virus in that they spread readily within a flock.
Prevention

- This allows for administration per os to a small percentage of the birds in a flock, which then spread infection to others, although this method is generally unsatisfactory for birds in wire cages.
- Vaccination by wing-web inoculation of AEV is also practiced in many flocks, but this method may carry some risk of clinical signs.
- Generally, vaccination is done after 8 weeks of age and at least 4 weeks before egg production.
Prevention

• It is very important that embryo adaptation of strains used for live virus vaccines does not occur because:
  • 1) adapted virus loses its ability to infect via the intestinal tract and is, therefore, no longer efficacious when administered by naturally occurring routes.
  • 2) adapted virus, like field strains, can cause clinical disease when administered by the wing-web route.
• Adaptation is detected by careful monitoring of inoculated embryos used in the production of vaccine for characteristic signs and any adapted virus can be eliminated from vaccine seed virus stocks by passage in susceptible chicks inoculated orally.
THANK YOU