AVIAN ENCEPHALOMYELITIS
(AE; Epidemic Tremor)

DEFINITION

Avian encephalomyelitis (AE) is a viral infection of chickens, turkeys, pheasants, and coturnix quail characterized in young birds by ataxia progressing to paralysis and, usually, by tremors of the head and neck. Infected adults usually show no signs.

OCCURRENCE

Clinical outbreaks are usually observed in chickens and most outbreaks are in 1-3-week-old chicks. Turkey poults, pheasants, and coturnix quail are also infected naturally. Experimental infection has been induced in ducklings, guinea fowl, and pigeon hatchlings. Infection can occur in older birds but usually is clinically inapparent. AE is worldwide in distribution.

HISTORICAL INFORMATION

1. In 1930 AE was first seen in 2-week-old Rhode Island Red commercial chicks. Within a few years the disease was present in most of the other New England states and was referred to as "New England disease". Between 1955 and 1970 the disease was described successively in coturnix quail, pheasants, and turkeys.

2. A nationwide testing program for AE antibody revealed that many chicken flocks in the United States have antibody to AE virus.

3. Hatcheries once replaced baby chicks that had AE or developed AE shortly after delivery. This practice caused considerable loss to the hatcheries. Vaccination of the breeders was first successfully implemented in the 1950s and AE largely became controlled in commercial flocks by the 1960s.

ETIOLOGY

1. AE is caused by a hepatovirus belonging to the Picornaviridae family. There appear to be no serologic differences among isolates although they vary in their tissue tropisms. All field strains are enterotropic but some strains are more neurotropic than others and pathogenicity varies.

2. The virus can be grown in the yolk sac of chick embryos free of maternal antibodies and in a variety of tissue culture systems. Embryo-adapted strains are not infectious by the oral route, are highly neurotropic, and cause muscular dystrophy in inoculated embryos.

3. Virus is present in the feces of infected birds and will survive there for at least 4 weeks.

4. The virus is fairly resistant to various environmental conditions.

EPIDEMIOLOGY

1. During the acute phase of infection in laying chickens, a period up to 1 month, some layers shed virus in some of the eggs they lay. Although vertically transmitted AE may affect hatchability, many of the chicks will hatch and can show clinical signs of the disease as early as the 1st day of age. The infected chicks will eliminate virus in their feces and virus may spread horizontally to other chicks of the hatch. Younger chicks shed for longer a longer period of time than older chicks.
2. The method of transmission of AE to susceptible adult flocks is unknown but is probably via fomites. Multiage farms are more likely to be infected than those with single age groups.

CLINICAL SIGNS

1. In chicks, signs may be present at the time of hatch but usually occur between the 1st and 2nd week. Age resistance is marked if exposure is after 2-3 weeks of age.

2. In chicks, signs include dull expression, ataxia progressing to paralysis and prostration [Fig. 1; Avian encephalomyelitis; Cornell U], and tremors of the head and neck. Tremor may be inapparent but often can be accentuated if the bird is frightened or held inverted in the hand. Prostrate birds are soon trampled and killed by the other birds.

3. The morbidity in chicks is quite variable but may go as high as 60%. If most chicks in the flock come from immune dams, morbidity is usually low. Mortality averages 25%. Few birds with signs recover completely. Those that survive often fail to grow or produce eggs normally. Many survivors later develop a bluish opacity to the lens and have impaired vision [Fig. 2; Avian encephalomyelitis; Cornell U].

4. Layers seldom show signs when infection is going through the flock. However, good production records often reveal a significant decline in egg production generally lasting no more than 2 weeks.

LESIONS

1. Generally, there are no gross lesions. In chicks, whitish areas in musculature of the ventriculus can sometimes be observed. No gross lesions are seen in adult birds.

2. Microscopic lesions, if typical, have special diagnostic value. There is a disseminated, nonpurulent encephalomyelitis with widespread and marked perivascular cuffing. Two microscopic changes are especially helpful: swelling and chromatolysis of neurons [Fig. 3; Avian encephalomyelitis; NCSU] in nuclei (nucleus rotundus and nucleus ovoidalis) in the midbrain and cerebellum, and dense lymphoid aggregates in the muscle of the proventriculus and/or gizzard.

DIAGNOSIS

1. In chicks the history, age of the birds, and typical signs of central nervous system (CNS) lesions permit a strong presumptive diagnosis. The diagnosis can often be strengthened by histopathologic examination. Alternatively, the direct fluorescent antibody technique can be used to demonstrate AE viral antigen in infected chicks.

2. Isolation and identification of the virus from the brains of infected chicks is possible but is time consuming and expensive. Also, there must be a source of susceptible chick embryos and this usually necessitates a layer flock that has never been exposed to AE.

3. Antibodies to AE can be detected as early as 4 days postinfection and persist for at least 28 months. Serologic assays include the ELISA, immunodiffusion test, virus neutralization test, passive hemagglutinin test and the indirect FA test. Rising titers in sequential samples are highly suggestive of active infection.

4. AE must be differentiated from other diseases that cause signs of CNS disease in young birds. These include:

   - Newcastle disease
   - Arboviral infection
   - Vitamin deficiencies (E, A and Riboflavin)
   - Equine Encephalomyelitis Virus
   - Mycotic encephalitis
   - Brain abscesses
   - Marek's disease
   - Toxics
   - (salt, some pesticides, etc.)
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CONTROL

1. Chicks from immune hens are usually protected by parental immunity during the critical first few weeks after hatching. Breeding flocks can be vaccinated to provide maximum protection to their chicks. Although vaccination is usually conducted prior to the onset of lay, some killed vaccines can be used during production.

2. Both killed and live vaccines are used for vaccination and are effective. Live virus vaccines must not be embryo adapted as they lose their ability to infect orally and can cause clinical disease when administered parenterally. Live vaccine is given by the wing web stick method in combination with pox, via the drinking water, or by spray. Birds that will serve as breeders should not be vaccinated until they are at least 8 weeks old. One vaccination is usually adequate for the life of the bird. Live vaccines should be applied at least 4 weeks prior to production; vaccines used in chickens can be protective for turkeys.

3. Chicks from flocks that have been naturally infected will probably receive enough parental immunity so that they will not develop the disease.

TREATMENT

Treatment is of no value.