AVIAN ENCEPHALOMYELITIS

SLIDE STUDY SET #5

A CONTINUING EDUCATION PROGRAM PREPARED

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IN COOPERATION WITH THE

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OF

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Avian encephalomyelitis (AE) is a viral disease of chickens, turkeys, pheasants and coturnix quail. The infection in growing or adult birds is asymptomatic except for a temporary drop in egg production in mature females. In young birds up to about 5 weeks of age, the disease is characterized by progressive ataxia due to muscular incoordination. Sometimes a fine tremor of the head or neck can be elicited; thus, the popular term "epidemic tremor". Severely affected chicks usually die from failure to eat and drink and from trampling by penmates. Survivors may become blind from cataracts.

AE was first described in the United States in 1932. It is world-wide in distribution. Nearly all flocks eventually become infected.

The etiologic agent is an enterovirus (picornavirus group). It is shed in feces during the active period of infection and birds are readily infected by the oral route. The virus can survive in the environment for long periods. Tracking and fomites are probable means of spread from flock to flock.

Adult birds often drop in egg production (about 10%) for 1 to 2 weeks commencing 5 or 6 days after exposure. During this period, a variable number of eggs may be infected and these can hatch as

infected chicks. These congenitally infected chicks shed virus to hatchmates in the incubator and to broodermates. They develop clinical signs during the first few days, always by 7 days of age.

Susceptible chicks hatched or raised with infected chicks become infected by contact and develop clinical signs within 10 to about 16 days. Chicks exposed after 3 or more weeks of age do not develop neurologic signs but do have characteristic histopathologic lesions. Chicks with maternal antibody are protected.

Clinical signs in young chicks consist of dullness, and variable degrees of incoordination which becomes more pronounced when the chicks are forced to move. Often an affected chick will run normally for several steps and then falter and go down. In more advanced stages of paresis, the chicks lay on their side unable to stand. The fine tremors of the head and neck are not seen in all chicks and may often be elicited by subjecting a chick to a quick sharp motion.

Gross pathology is minimal. The gizzard musculature may have pale areas from necrosis and mononuclear cell infiltration and hydrocephalus may be seen. Cataract formation with opacity or bluish discoloration of the lens occurs in as many as 40% of the survivors in an AE outbreak.

Microscopic pathology is observed in the central nervous system (CNS) and in various visceral organs. Neuronal degeneration can be found in all areas of the CNS, and especially in the pons, medulla and anterior horn cells of the spinal cord. Purkinje cell degeneration is a characteristic change often seen in the

cerebellum. Glial foci are commonly found in the molecular layer of the cerebellum and perivascular cuffs of infiltrating small lymphocytes occur in all portions of the CNS. Foci of infiltrating small lymphocytes may also be seen in the dorsal root ganglia and a variety of visceral organs including the ventriculus, pancreas, heart, liver, and muscular layer of the proventriculus. Some lymphoid cell aggregates may normally be found in these visceral organs. However, the foci in AE are often composed of very dense aggregates and are unusual in the extent of their distribution as well as their size.

A permanent immunity to AE develops within 10 to 14 days in immunologically competent birds, i.e., after 3-4 weeks of age. Both precipitin and neutralizing antibody are elicited. Passive antibody in the yolk renders fertile eggs resistant to challenge with embryo-adapted (Van Roekel Strain) AE virus. This permits the use of an embryo susceptibility test to determine if breeding flocks are immune. Embryo-adapted virus is injected into the yolk sac of 5- to 6-day-old embryos which are examined 10 to 12 days later for evidence of paralysis and muscular dystrophy. If the flock is immune, the embryos remain normal.

Diagnosis of AE in clinically affected chicks can be made by virus isolation, histopathology or fluorescent antibody tests. Virus isolation is best performed by inoculating a suspension of brain material into the yolk sacs of 5- to 6-day old embryos from a known susceptible flock. The eggs are hatched and the chicks observed for characteristic signs of AE during the first 7 to 10

days of life. Histopathologic diagnosis is best performed on affected chicks which are at least 10 or 11 days old and is based on finding characteristic lesions in the CNS and in visceral organs such as proventriculus, pancreas and ventriculus. Fluorescent antibody tests conducted on brain smears can give a definitive diagnosis but negative tests should not be considered proof that AE is not the proper diagnosis.

When AE is suspected in older birds (e.g. in a flock experiencing a drop in egg production), serologic tests are suitable for a diagnosis. Virus neutralization tests with embryo-adapted virus are conducted on paired acute and convalescent sera. A rise in antibody titer is diagnostic. The average neutralization index for a negative flock should be around 0 while that for a positive flock should be greater than 1.0. If fertile eggs are available, embryo susceptibility tests can be conducted during the acute and convalescent periods to demonstrate a shift from susceptible to resistant status. Either test requires eggs from a known susceptible flock (for the test itself or as a control).

A differential diagnosis must consider other neurologic disorders such as encephalomalacia, Newcastle disease, Marek's disease and equine encephalomyelitis. AE usually can be distinguished on the basis of age (less than 5 weeks), general absence of gross lesions, and characteristic histopathology, i.e. gliosis, neuronal and Purkinje cell degeneration, perivascular cuffing and hyperplastic lymphoid follicles in certain visceral organs. A definitive diagnosis requires virologic or serologic

investigations.

Clinical disease is prevented by vaccination after 8 weeks of age and at least one month before egg production. A live virus vaccine is administered by the oral route. Inactivated vaccines are useful in certain instances.

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SLIDE SET DESCRIPTIONS

1. Clinical signs consist of progressive ataxia, weakness, dullness and sometimes a fine tremor of the head and neck. Slight incoordination can be elicited by forcing the chicks to move. Reluctance or inability to stand (as seen in these chicks) is common.
2. Gizzard musculature may have grossly visible pale areas resulting from massive infiltration by mononuclear cells. (Slide by M.C. Peckham, Cornell University, Ithaca, NY).

3 and 4. Compact and diffuse microgliosis is a characteristic lesion in cerebellum, particularly in the white matter (slide 3) or molecular layer (slide 4). (H&E; 125x).

5. Perivascular cuffs of small lymphocytes, often several layers thick are found in nearly all parts of the CNS. (H&E: 200x).

6 and 7. Neuronal degeneration is considered one of the most striking features of AE. The normal neurones seen in slide 6 (brain stem; H&E; 200x) contrast with an affected one undergoing central chromatolysis seen in slide 7 (cerebellum; H&E; 400x)

8 and 9. Similarly, Purkinje cells may undergo necrosis, and some areas of the cerebellum are often found to be essentially devoid of these cells. The normal cerebellum (slide 8) has a uniform row of Purkinje cells separating the granular and molecular layers. In contrast, few such cells are seen in a portion of a cerebellar folium from a

clinically affected chick (slide 9). Purkinje cells have been found to be a frequent site of viral antigen when examined by the fluorescent antibody test. (Both slides H&E; 100x).

10. The muscular layer in this proventriculus is infiltrated by dense aggregates of lymphocytes. A lesion like this, perhaps coupled with abnormal aggregates in the gizzard and pancreas, is of diagnostic significance in AE only when accompanied by characteristic microscopic lesions in the CNS. (H&E; 100x).
11. Gizzard musculature is frequently infiltrated in the same manner as the proventricular musculature. (H&E; 100x).
12. Infiltration of the myocardium is frequent in AE but because some lymphoid cell aggregates are found in "normal" hearts, caution is required in interpretation. In this specimen, abnormal numbers of cells infiltrate between the muscle fibers. (H&E; 200x).
13. As in the heart, normal lymphoid foci appear in the pancreas. In AE there is an increase in their number and size. Several such foci are seen in this pancreas. (H&E; 100x).
14. A lymphoid focus in the liver of an infected chick. The comments regarding the heart and pancreas also pertain to the liver. (H&E; 100x).
15. Susceptible (antibody free) embryos, inoculated with embryo-adapted AE virus via the yolk sac at 5 or 6 days of incubation, develop lesions illustrated by the 2 embryos on the left. Severe stunting results primarily from marked

muscular dystrophy, as seen in the embryo from which the skin has been removed. In addition, affected embryos are paralyzed and the legs are rigidly extended rather than hanging loosely from the body. The 2 embryos on the right are normal. Because embryos from immune flocks are resistant to infection, an "embryo susceptibility" test can be used to determine if a breeding flock has been exposed to AE.

16. Cataract formation as seen in the affected eye on the left may occur in a high percentage of the survivors of an early outbreak of AE. The lesion is usually seen as an opacity or bluish discoloration of one or both eyes. (Slide by M.C. Peckham, Cornell University, Ithaca, NY).