CORONAVIRAL ENTERITIS OF TURKEYS
(Transmissible Enteritis of Turkeys; Blue Comb)

DEFINITION

Coronaviral enteritis is an acute, highly infectious disease of turkeys, especially poults, characterized by anorexia, diarrhea, dehydration, and variable mortality.

OCCURRENCE

Coronaviral enteritis occurs in turkeys of all ages but is seen more frequently in young turkeys. Chickens are not infected. The disease occurs throughout the year. It is recognized in the United States, Canada, and Australia; however, the incidence has substantially decreased since the 1970s.

HISTORICAL INFORMATION

1. Coronaviral enteritis was first reported in 1951 in Washington and shortly thereafter in Minnesota. The disease has not been reported frequently from other states.

2. For many years this disease was reported as the most costly disease of the turkey industry in Minnesota. There it accounted for 23% of total turkey mortality in 1966. The last reported outbreak in Minnesota occurred in 1977.

3. Transmissible enteritis is distinct from hemorrhagic enteritis, another disease of turkeys.

ETIOLOGY

1. A coronavirus is now accepted as the cause of transmissible enteritis. There is no relationship between this virus and other avian or mammalian coronaviruses. In the past the disease has been attributed to many different agents, including vibrio and a variety of viruses.

2. Under experimental conditions the virus is readily destroyed in batteries and cages. Destruction of the virus probably is more difficult under natural conditions because it survives well in frozen feces. Under field conditions, cleaning and disinfection of buildings and equipment, combined with at least 3-4 weeks of depopulation, is advisable. Virus survival in ranges can be extensive despite depopulation.

3. A number of other enteric viruses including rotavirus, reovirus, astrovirus, enterovirus, and calicivirus have been identified from turkey feces. Their role in enteritis is uncertain.

EPIZOOTIOLOGY

The virus is shed in the feces of recovered carrier birds for several months. Further, the virus persists in frozen feces for several months. The virus can be spread by contact of susceptible birds with infected birds or their feces. Once introduced into a flock, the disease spreads rapidly among susceptible birds. There is no evidence that the virus is transmitted through the egg.

CLINICAL SIGNS

1. In young poults the signs appear suddenly after an incubation period of 1-5 days. Signs include anorexia, depression, frothy diarrhea, subnormal temperatures, darkening of the head and skin, and loss of weight. Birds tend to huddle around heat sources. Spread is rapid and morbidity is close to 100%.

2. The signs seen in young poults may also be observed in laying turkeys but usually are less marked. Moreover, there is a sudden decrease in egg production and some eggshells are chalky.
3. Good husbandry and supplemental heat tend to suppress mortality. Mortality varies with the age of the birds affected and can range from 5 to 50% in natural infections.

4. The course of the disease in a flock is around 2 weeks. Recovery may be prolonged, particularly in males, and the flock may become uneven in size.

LESIONS

1. There is marked dehydration and emaciation, especially in older birds.

2. Enteritis [Fig.1; Coronaviral enteritis; Cornell U] is a consistent lesion and there may be petechial hemorrhages in the mucosa. Watery, gaseous ingesta with excess mucus and occasional casts are often observed. Ceca are distended with odiferous, watery contents.

3. The pancreas has a whitish, chalky appearance. This change is not entirely uniform but is usually present to some degree.

4. Excessive urates are frequently found in the kidneys and ureters. The spleen often is small.

5. Microscopically, the villus-to-crypt ratio is decreased, particularly in the jejunum. There are large numbers of round cells in the adrenals. In the pancreas there is focal hydropic degeneration and some cells contain large eosinophilic intracytoplasmic inclusions, which are aggregates of intracytoplasmic granules.

DIAGNOSIS

1. The history, signs, and lesions are suggestive of the diagnosis but are duplicated by certain other diseases. Filtered intestinal material can be given to a few susceptible poults and will reproduce the disease in a few days.

2. A direct fluorescent antibody (FA) technique can be used to identify the virus in the cytoplasm of intestinal mucosal cells and in the bursa. An indirect FA technique can be used to detect antibodies in the serum 14 days or more after exposure to the virus.

3. The virus can be isolated in embryonating turkey eggs and can be identified by electron microscopy (EM). EM can also be used to detect virus particles in intestinal contents.

4. In differential diagnosis one should consider the following diseases:

   - Young Poults (Less than 7 Weeks)
   - Salmonellosis
   - Hexamitiasis
   - Starve outs (very young birds only)
   - Coccidiosis
   - Water deprivation

   - Growing and Mature Turkeys
   - Fowl cholera
   - Erysipelas
   - Blackhead
   - Trichomoniasis
   - Hemorrhagic enteritis

CONTROL

1. Turkeys should be reared under the all-in, all-out system. Use quarantine and a high standard of sanitation to prevent introduction of the virus.

2. If the disease has been present in prior broods, thoroughly clean and disinfect the premises after complete depopulation. Leave the premises empty for at least a month, perhaps longer during the winter season.

3. No vaccine is available for immunization.
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TREATMENT

1. Treatment is largely empirical. Broad-spectrum antibiotics often appear to suppress mortality by preventing secondary infections. Therapeutic agents that have been reported to be of value include penicillin, streptomycin, tetracyclines, bacitracin, and neomycin.

2. Calf milk replacer (25 lb/100 gal) or potassium chloride (450 g/100 gal) may be added to the drinking water for a few days. In mature birds molasses is sometimes added to the drinking water (1 pint/5 gal) for a 1-day flush before administration of a course of antibiotics.