CHICKEN INFECTIOUS ANEMIA
(CIA; Chicken Anemia Virus; Blue Wing Disease)

DEFINITION

Chicken infectious anemia (CIA) is a disease of chickens caused by a circovirus and characterized by aplastic anemia, generalized lymphoid atrophy, subcutaneous and intramuscular hemorrhage, and immunodepression.

OCCURRENCE

CIA is probably ubiquitous in all major chicken-producing countries in the world.

HISTORICAL INFORMATION

CIA virus was first isolated by Yuasa in Japan in 1979. It has also been called chicken anemia agent, chicken anemia virus, and parvovirus-like virus. The clinical signs and lesions previously described as blue wing disease, anemia-dermatitis syndrome, and hemorrhagic anemia may have been caused by CIA virus.

ETIOLOGY

1. CIA virus is the only member of the genus Gyrovirus of the Circoviridae.
2. CIA virus is difficult to isolate due to the restricted cell lines suitable for propagation. Most chicken embryo cell lines and chick embryos are resistant to infection or produce low virus yields.
3. Bioassay in susceptible 1-day-old chicks is the most specific method for primary isolation.
4. CIA virus is extremely resistant.
5. No antigenic differences have been noted amongst strains.

EPIDEMIOLOGY

1. CIA virus is thought to be ubiquitous in poultry-producing areas of the world.
2. Chickens are the only known hosts.
3. All ages are susceptible to infection but clinical disease is seen only during the first 2 to 4 weeks. However, age resistance is delayed by simultaneous infection with infectious bursal disease virus.
4. The most important method of transmission is vertical from infected hens. Antibody-negative chicks are most susceptible to clinical disease. CIA virus also easily spreads via feces among birds in a population.

CLINICAL SIGNS

1. The only specific sign of CIA is anemia characterized by hematocrit values ranging from 6 to 27% (normal hematocrit value is 35%)
2. Nonspecific clinical signs include depression, pale tissues, depressed weight gain, and secondary bacterial, mycotic, and viral infections.
3. Morbidity and mortality rates vary depending upon altered immune status due to other infections such as infectious bursal disease, Marek’s disease, or reticuloendotheliosis. Mortality is usually 5-10% but can be 60%.

4. Early infections with CIA virus can interfere with vaccination against Marek’s disease or infectious bursal disease.

LESIONS

1. Thymic atrophy is the most consistent lesion.

2. Fatty yellowish bone marrow, particularly in the femur, is characteristic [Fig. 1; Chicken infectious anemia; Cornell U].

3. Bursal atrophy can also be seen.

4. Hemorrhages in the mucosa of the proventriculus, subcutis, and muscles and a swollen mottled liver can also be observed.

5. Lesions of secondary bacterial infections, such as gangrenous dermatitis and blue wing disease can be seen in commercial flocks.

DIAGNOSIS

1. A presumptive diagnosis is based upon clinical signs and gross lesions.

2. Isolation and identification of the virus from most tissues, buffy coat cells, and cloacal contents.

3. Serologic assays to detect antibodies such as the ELISA, virus neutralization test, and indirect immunofluorescence.

4. PCR is the test of choice for identification of CIA virus in cell cultures and chicken tissues.

CONTROL

1. Best prevention is by immunization of breeder flocks prior to the onset of egg production (between 13-15 weeks of age but no closer to egg production than 4 weeks).

2. Where vaccines are not available, nonimmune breeder flocks are exposed to the virus during the growing period by rearing them on litter transferred from previously infected breeder houses. This is an effective but obviously risky practice.

TREATMENT

1. There is no satisfactory treatment for the viral infection.

2. Use supplemental vitamins via drinking water, including B vitamins, and A, E, and K.

3. Treat secondary gangrenous dermatitis with penicillin, bacitracin, lincomycin, or other antibiotics effective against Clostridium spp. or Staphylococcus spp.