CHICKEN INFECTIOUS ANEMIA

Slide study set # 20

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By: H. L. SHIVAPRASAD and B. LUCIO

Chicken infectious anemia (CIA) is a disease characterized by aplastic anemia, generalized lymphoid depletion, subcutaneous and intramuscular hemorrhages, and immunodepression. Because of the immunodepression, increased mortality due to secondary complications is often observed. The causative agent of CIA, first identified in Japan in 1979, has been called at various times, chicken anemia agent (CAA), parvovirus-like virus (PVLV), chicken anemia virus (CAV), or chicken infectious anemia virus (CIAV). Clinical signs and lesions described previously in cases of aplastic anemia, hemorrhagic syndrome, and anemia-dermatitis may have been caused by CIAV.

Incidence and susceptible hosts. Chicken infectious anemia virus is ubiquitous in all major chicken producing countries of the world. Serological data indicate that the virus is widely spread in the United States in both layer and broiler-type breeder chickens. The chicken appears to be the only host for CIAV.

Transmission. The disease readily spreads horizontally, but vertical transmission appears to be the most important means of dissemination. Vertical transmission occurs following the infection of hens in lay. The hen continues transmitting CIAV until antibodies appear in her blood, a period of approximately 7 days. From a single infected breeder flock, however, CIAV infected chicks can be produced for 3 to 6 weeks.

Pathogenesis. Chickens of all ages are susceptible to infection with CIAV. However, clinical disease is seen only during the first two to three weeks of life although immunocompromised chickens may suffer from anemia later in life. Chicken infectious anemia virus persists only for 3 to 4 weeks in chickens with an intact immune system, but for as long as 7 weeks in immunocompromised chicks.

Age resistance to clinical disease caused by CIAV develops rapidly and becomes complete by 2 to 3 weeks of age. Maternal antibodies from immune hens prevent clinical disease in young chicks. Because of passive immunity and age resistance, most infections with CIAV are subclinical.

Etiology. Chicken infectious anemia virus is a spherical, nonenveloped virus with a diameter of 19 to 24 nm. The genome consists of a circular, single-stranded molecule of DNA. Two other viruses with similar characteristics are the psittacine beak and feather disease virus (PBFDV), and swine circovirus (SC). However, antigenic and nucleic acid sequence studies have detected no relationship among them. It has been suggested that these three viruses should be placed in a new viral family, Circoviridae. Chicken infectious anemia virus is a remarkably hardy virus and is resistant to treatment for 2 hours at 37 C with 5% solutions of invert soap, amphoteric soap, ortho-dichlorobenzene, iodine disinfectants, and sodium hypochlorite. It is completely

inactivated by treatment with 10% iodine disinfectants and sodium hypochlorite in 2 hrs at 37 C. Heating at 100 C but not at 80 C for 15 minutes destroys CIAV infectivity.

Clinical signs. The only specific clinical sign of CIA is anemia, which reaches a peak at 14 to 16 days post infection. At this time, hematocrit values can range from 6 to 27% (normal 35%). Levels of thrombocytes and white blood cells are reduced and the blood may be slow to clot. Affected birds appear depressed and pale, and may show signs of secondary bacterial, fungal or viral infections. Morbidity and mortality rates are influenced by several factors such as immunodepression by other agents [infectious bursal disease virus (IBDV) and Marek's disease virus (MDV), among others], secondary infections, age of infection, route of infection, and environmental factors. Mortality is usually between 1 and 10%, but can be as high as 60%. Morbidity and mortality are severe if chicks are dually infected with CIAV and MDV, reticuloendotheliosis virus (REV) or IBDV. Infection of chicks with CIAV in the early part of life can interfere with vaccination against MDV or IBDV.

Gross lesions. Bone marrow of the femur can be fatty and yellowish due to severe anemia. Moderate to severe thymic atrophy is one of the most consistent lesions seen in chicks affected with CIA. Atrophy of the bursa of Fabricius can also be seen. Enlarged and mottled livers, hemorrhages in the proventricular mucosa, and subcutaneous and muscular hemorrhages are often seen in association with severe anemia. Secondary bacterial infections can cause airsacculitis, pericarditis, pneumonia, etc. Gangrenous dermatitis resulting from secondary bacterial infections is often observed. Skin lesions usually develop on the wings as seen in "blue wing disease II, but may also appear on other parts of the body.

Microscopic lesions. Hypoplasia of both erythroid and myeloid series cells is seen in the bone marrow by 8 days post-infection. Hematopoietic cells are replaced by adipose tissue or proliferating stromal cells in the early stages of the disease. Immature erythrocytes and granulocytes are present in increasing numbers by 20 days post-infection, and there is a period of hyperplasia by 20 to 24 days post-infection. The

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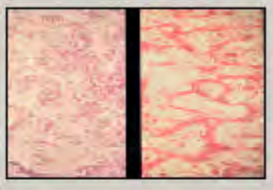
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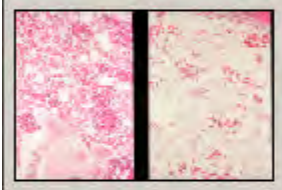
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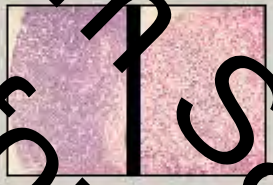
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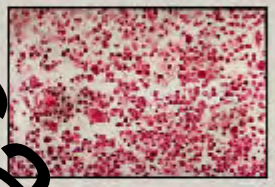
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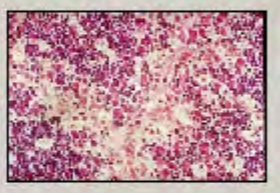
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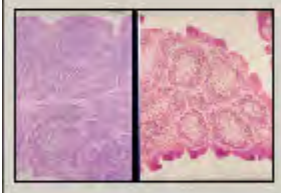
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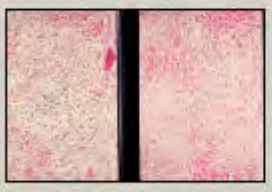
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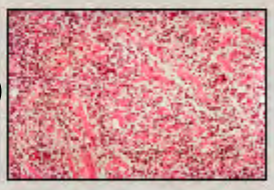
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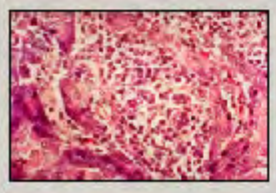
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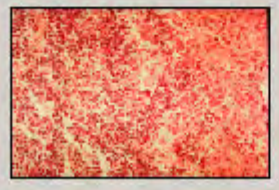
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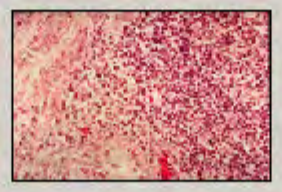
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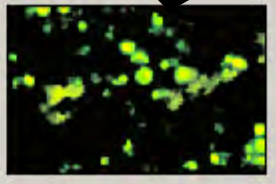
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