CHICKEN INFECTIOUS ANEMIA

Slide study set # 20

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By: J.A. Smyth, H. L. Shivaprasad and K.A. Schat

Chicken infectious anemia (CIA) is a disease characterized by aplastic anemia, thymic atrophy, subcutaneous and intramuscular hemorrhages, and immunosuppression. Because of subclinical immunosuppression, increased mortality due to secondary infections such as gangrenous dermatitis and fungal pneumonia is often observed. Chicken infectious anemia virus (CIAV) or (CAV), first described in Japan in 1979, has been found in virtual all commercial and backyard chicken flocks. 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 (CIAV) is ubiquitous in all major chicken producing countries of the world. Serological data indicate that the virus is wide-spread in the United States in broiler, layer and breeder flocks of chickens and was already present in the USA in the 1950's. Chickens are the major host for CIAV.

Transmission. CIAV spreads horizontally mostly by the fecal oral route although feathers can also be a source of infection. In addition, vertical transmission occurs following horizontal infection of hens lacking virus neutralizing (VN) antibodies. Transmission by virus-positive semen has also been reported. Viremic hens continue transmitting CIAV until the development of VN antibodies. CIAV-positive chicks may be produced for 3 to 6 weeks once an antibody-negative breeder flock becomes infected.

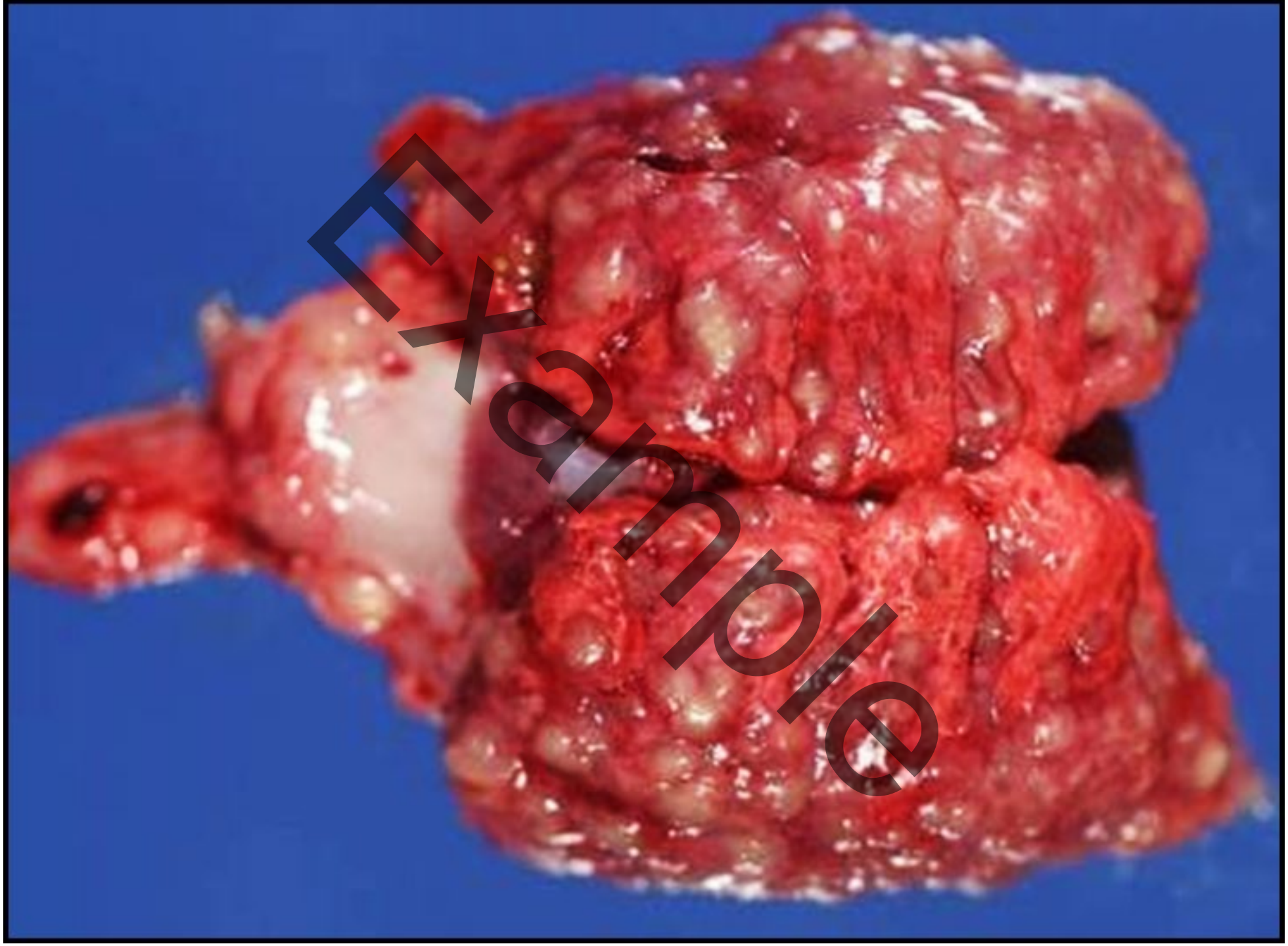
Pathogenesis. Chickens of all ages are susceptible to infection with CIAV. However, clinical disease is usually seen only during the first two to three weeks of life. Older chickens may develop anemia if immunocompromised at the time of infection. In chickens with an intact immune system, virus replication persists until VN antibodies develop. Although virus replication will cease, CIAV can remain present as latent virus in many organs including the gonads. Virus replication can be prolonged in immunocompromised chickens, especially if the humoral immune system is compromised by e.g., infectious bursal disease virus (IBDV).

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 maternally derived antibodies and age resistance, most infections with CIAV are subclinical.

Etiology. CIAV has been classified as the only virus species in the genus Gyrovirus in the Gyrovirinae subfamily of the Anelloviridae family. CIAV is an icosahedral, non-enveloped virus with a diameter of 25 nm and a genome of 2.3 kb nucleotides consisting of circular, single-stranded, negative sense DNA. During virus replication a single mRNA is generated resulting in the production of 3 viral proteins. VP1 is the only viral protein present in the virus particles, while VP2 is needed to properly fold VP1. VP3, also known as apoptin, is the major cause of apoptosis of infected cells, although VP2 may also cause apoptosis. There are several genotypes of CIAV but there is only one serotype.

CIAV 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. CIAV is also resistant to fumigation with formaldehyde. CIAV is inactivated by treatment with


Example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