INCLUSION BODY HEPATITIS IN BROILERS: EVOLUTION OF THE LESIONS

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SUMMARY. Twenty day old broilers (n= 8) with viral hepatitis suspected were necropsied. All animals show ascites and pale mottled reddish livers. Lymphoid tissues (thymus and bursa of Fabricius) were atrophied. Microscopic study revealed subacute hepatitis with multifocal necrosis and presence of inclusion bodies by adenovirus. Bursa displayed a severe and extensive necrosis of lymphoid cells in four animals. Histological findings suggested the final diagnosis of Inclusion Body Hepatitis (IBH). Additional samples from 8 birds (liver, spleen, bursa and tracheae) at 42 day old, from the same flock were submitted. Pathological study revealed severe atrophy of the bursa of Fabricius with marked depletion of lymphoid tissue and chronic hepatitis in 4/8 livers (inclusion bodies are not appreciated). We discussed the infection by adenovirus in chickens as a primary pathogen; however it can also be secondary with other infectious agents, such as virus of Gumboro Disease, caused a severe immunosuppression of animals.

CASE REPORT
The disease Inclusion Body Hepatitis (IBH) is an adenovirus infection described in young chickens, from 4 up 8 weeks. Macroscopically, this disease is characterized by hepatomegaly, ascites and the presence of white or reddish multifocal areas (mottled). Histologically, multifocal necrosis of hepatocytes with inflammation is observed. Basophilic intranuclear inclusion bodies are often seen in the cellular elements around the necrotic focus. Chicken infection could be primary or secondary to an immunosuppression process due to other previous viral infections like Gumboro disease or infectious anaemia, playing a role of secondary pathogen in these cases.

Eight broilers of twenty days-old with viral hepatitis suspected are received in the Pathology Service. Significant mortality at 17-18 days of age were observed, reaching
10% of the flock. At necropsy, all animals displayed ascites, pale kidneys, yellowish livers with mottled reddish foci, as well as a bursa of Fabricius and thymus atrophy. A subacute hepatitis with multifocal necrosis and presence of inclusion bodies by adenovirus in all animals were observed under a microscope. Marked necrosis of lymphoid tissue were observed in the bursa of the four animals. Therefore, the final diagnosis was a viral inclusion body hepatitis (adenovirus), possibly as consequence of a severe immunosuppression due to Gumboro virus. Follow-up 42 days marked a high mortality, reaching 22% of animals (of a total of 33,000 animals, almost 7000 die to infectious process). Additional samples from livers, spleens, bursa and tracheae fixed in formalin were submitted for histological examination. Microscopical diagnosis showed: bursa of Fabricius with marked depletion of lymphoid cells and severe atrophy. Chronic hepatitis in 4/8 livers (inclusion bodies are not appreciated). Mild nonspecific diffuse tracheitis. Our case described typical lesions of a severe immunodeficiency (suspect of Gumboro disease) with chronic course. Chronic hepatitis in some animals could be the result of the evolution of the infection by adenovirus (no intranuclear inclusion bodies were observed). Final conclusion is considered that adenovirus induced of hepatitis in chicken as primary pathogen, and can also be secondary or act associated with other agents, such as Gumboro virus, giving place to clinical pictures with severe immunosuppression in birds.

REFERENCES

