CASE OF INTEREST

A Case of Inclusion Body Hepatitis in a Flock of Broiler Chickens.
By Richard Fox, Veterinary Pathologist.
Case from Judith Hargreaves.

A broiler farm presented the visiting veterinarian with an acute onset of mortality in a flock of 12 week old broiler chickens. Initial post-mortem examination revealed only multifocal areas of pallor affecting liver tissue and areas of soft pallor of the pancreas in several birds. Tissue samples were taken for histopathology of all major visceral organs.

Histologically the samples of intestine, kidney and spleen were all normal in appearance. Two sections of liver displayed extensive hepatocellular necrosis (Figure 1). Inflammatory infiltrates within the liver tissue were mild except for some mixed cellular infiltrates around some of the larger vessels. There were numerous large basophilic intranuclear inclusion bodies visible within hepatocytes primarily bordering areas of necrosis (Figure 2). There was also bile duct proliferation around portal areas.

One bursa of fabricious was submitted which displayed severe lymphoid atrophy with focal granulocyte infiltration. Portions of pancreas attached to the intestine also displayed multifocal areas of acinar necrosis with an associated cellular infiltrate and overlying serositis. Inclusion bodies are not evident. There is one bursa present. This shows severe lymphoid atrophy with focal granulocyte infiltration.

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of the broilers and the histopathologic changes, was made.

IBH, a fowl adenovirus Group 1 infection, can affect all ages of broilers and all chickens are found to be susceptible even in immunological intact chicks during the first 2-3 weeks of life. Chicks as young as 5 days of age can develop IBH. Recurring infections were noticed in 2 or 3 successive flocks on the same farm. Broilers are usually subject to varying degrees of immune challenge as a result of infection with Bursal Disease virus, CAV (Chicken Anemia virus), Marek's disease virus and during stress management. Role of the adenoviruses in these conditions was found to be a secondary pathogen. Reports of virulent strains without prior immune suppression are known. Recently some strains of serotype 4 fowl adenoviruses cause inclusion body hepatitis and hydropericardium syndrome in chickens.

The liver is the primary organ affected in these birds which is enlarged, pale yellow with multiple petechial haemorrhages. In some cases kidneys appear swollen and pale due to deposition of urates. Skin and body fat are yellow in colour (jaundice). Bursa and thymus appear smaller.

Diagnosis can be made initially on examination of liver tissue with the characteristic intranuclear basophilic inclusion bodies in hepatocytes. Virus isolation can be performed on Clocal swabs (faeces) and fresh liver with lesions. Bursae of Fabricius display atrophy of follicles, beginning with loss of medullary lymphoid cells and proceeding to a marked interlobular and intralobular fibrosis. Atrophic thymuses are characterized by loss of lymphocytes and hemorrhage. Pancreatic necrosis and ventricular erosions are also reported with IBH.

Virus isolation performed as the routine procedure for serotyping IBH virus, in embryonated eggs/cell culture and by electron microscopy. Serology can be used to monitor progression but obviously does not indicate active infection. Molecular level PCR & REA (Restriction Endonuclease Analysis) methods can also be used for detection and typing of field isolates.

Bibliography

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