Diagnostic Exercise
From The Davis-Thompson Foundation*

Case #: 127 Month: August Year: 2019

Answer Sheet

Title: Avian adenoviral infection in turkeys

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Microscopic findings:

Figure 1. Spleen. 400X, H&E.

Morphologic Diagnosis: Spleen: Marked reticuloendothelial hyperplasia, lymphoid atrophy and granulocytic splenitis, diffuse, severe, with intranuclear basophilic smudgy viral inclusions consistent with Aviadenovirus.
**Discussion**: The gross and histologic lesions are consistent with Turkey Hemorrhagic Enteritis due to Adenovirus. In addition, culture of the intestines yielded very heavy growth of Clostridium perfringens, though no toxin studies were conducted. In addition to the spleen, the small intestine had severe, diffuse, necrotizing lesions with intranuclear inclusion bodies and Peyer’s patch necrosis. The liver had mild, multifocal, hepatitis with hepatocellular necrosis and intranuclear inclusion bodies.

The histologic findings of splenitis with intranuclear smudgy basophilic viral inclusions that peripheralize the chromatin (Figure 2), hemorrhagic enteritis, and necrotizing hepatitis (not pictured) are consistent with Aviadenovirus, genus Siadenovirus, which primarily affects young chickens, ducks, turkeys, pheasants and quail. Viruses in this genus cause diseases such as Quail Bronchitis (QB), Egg Drop Syndrome (EDS), Turkey Hemorrhagic Enteritis (THE), Pheasant Marble Spleen Disease (MSD), Falcon adenovirus A infection, and Inclusion Body Hepatitis (IBH).

While adenoviruses affect mostly fowl, including water fowl, recently a novel strain of adenovirus was found in red-bellied parrots (*Poicephalus rufiventris*). This is the first case reported in psittacines, providing evidence of frequent host switch events and recombination among adenovirus progenitors in avian hosts. This new strain is called *Poicephalus adulavirus* (PoAdV). Adenoviruses have also been found in Gouldian finches. Raptor adenovirus-1 (RAvV-1) was identified as the causative agent of an outbreak of adenoviral disease in the United Kingdom in 2004.

Turkey adenovirus 3 (TAdV-3) is the most virulent strain in this avian species. One study found that outbreaks occurred at the age of 6 to 8 weeks and were complicated with secondary bacterial infections, primarily *Escherichia coli*. The reason for the outbreak occurring at this age is due to the decline of maternal antibody levels at 5-7 weeks of age. If the young birds do not succumb to viremia, antibodies then increase sharply due to natural infection with THEV. Turkeys are vaccinated at 5 weeks of age and within four weeks have high antibody titers.

Researchers have successfully mapped the structure of the fibre head of the avirulent and virulent variants of turkey adenovirus 3 (TAdV-3). This is important because avirulent strains are used to prepare live vaccinations. Based on the genomic map, the following characteristics may provide insight into the pathogenicity:

- A positively charged surface may be important for host cell interaction
- TadV-3 is the only strain that has a beta-hairpin insertion in the C-strand contacting a neighboring monomer
- The fibre head shares topology with other adenovirus and reovirus fibre heads and with certain bacteriophage baseplate protein receptor-binding domains
- The sialyl group of sialyllactose was identified as a possible receptor or coreceptor for the TAdV-3 fibre head
- A primary receptor may well be a more complex carbohydrate or a sialylated cell surface molecule, possibly a glycolipid in nature, with a more extensive interaction footprint on the fibre head,
Recent outbreaks among vaccinated turkeys in Germany suggest that genetic drift has resulted in less efficacy of immunization. This genetic drift suggests evolution of THEV, including virulent and vaccine-derived strains in the field, which may lead to evasion of vaccinal immunity by drifted viruses and/or increase in the virulence of field strains.¹ Knowledge of the structure and receptor-binding properties of the TAdV-3 fibre head may allow the design of chimeric adenoviruses, which in turn may lead to vaccination or gene therapy vectors that may target specific cell types.⁶ Until a cure is found, virulent outbreaks have been successfully treated and controlled by serum injections from recovered flocks.

References:


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