



Blue Wing Disease



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Abbreviations: BWD: Blue Wing Disease; CAV: Chicken Anaemia Virus; ARV: Avian Reovirus; SPF: Specific-Pathogen-Free; NDV-F: Newcastle Disease Virus-F; REV: Reticuloendotheliosis Virus; MDV: Marek's Disease Virus.

Mini Review

Blue wing disease (BWD) which occurs after co-infection of chicken anaemia virus (CAV) with avian reovirus (ARV) has been known in Sweden since 1972 [1]. Most of the outbreaks have affected young broilers between 2 and 4 weeks of age with a mortality rate of 1 to 60%. The most characteristic lesions are

subcutaneous and intramuscular haemorrhages, most frequently on the wings (Figure 1), as well as atrophy of thymus, spleen and bursa of Fabricius. A similar syndrome has been reported from Germany, Denmark, Great Britain, Poland, Belgium [2]. The skin lesions in the form of ecchymotic haemorrhages are often infected secondarily by bacteria leading to a gangrenous dermatitis [1].



Figure 1: Blue wing disease.

The lesions found in the Swedish cases of BWD [1] were very similar to those described in chickens, experimentally infected with CAV [3] but the subcutaneous haemorrhages were most pronounced and the anaemia less severe in BWD, and the atrophy of thymus and bursa of Fabricius was seen in both BWD and experimental CAV infections.

CAV and ARV are immunosuppressive chicken viruses which affect immune function in chickens and lead to immunosuppression

causing serious economic losses in the chicken industry because immunosuppressed chickens are more susceptible to viral and bacterial pathogens, respond poorly to vaccination, and display lower feed conversion efficiency as well as growth retardation [4]. The infection with CAV has been described in most countries with a developed chicken industry [5]. Reovirus infections are also prevalent worldwide in chickens and have been associated with many diseases in poultry of which viral arthritis/tenosynovitis is the most important [6].

Since the first description of BWD in Sweden in 1984 [1] many authors provided evidence of the results after experimental infection with CAV and ARV. Enström et al. [7] found that two weeks after inoculation of one-day-old specific-pathogen-free (SPF) chicks with isolates of CAV and ARV from a field case of BWD, several birds died or were killed when they became moribund. These birds had petechial haemorrhages in the skin, atrophy of the thymus and the bursa of Fabricius and also atrophic bone marrow. McNeilly et al. [8] found out that one-day-old SPF white leghorn chickens dually infected with CAV and ARV had, fourteen days after inoculation, significantly lower weight gain and more severe tissue damage than chicks inoculated with either virus alone. More severe and pronounced lesions in experimentally co-infected broiler chicks were also ascertained by Bhardwaj et al. [9] who made the conclusion that chickens infected at an early age with CAV and subsequently with ARV exhibited exaggerated and extended lesions along with suppressed NDV-F vaccine responses. Meng Bin et al. [10] reported that co-infection of CAV and ARV existed commonly in chicken flocks in Shandong province of China.

There is no specific treatment for infected birds with these viruses. Vertical spread of CAV can be controlled by the vaccination of breeding hens before or around point of lay that reduces the vertical transmission rate. At the current stage, there are intensive searches for new vaccines with enhanced immunogenicity and reduced pathogenic potential among strains with artificial induced mutations in progress [11-13]. Prevention of reovirus infections can be achieved by vaccination of broiler breeders with a combination of a live attenuated and inactivated vaccines to confer maternal immunity in progeny for early protection against field challenge. Current commercial vaccine strains have been used for decades to control diseases associated with reoviruses. In broilers, occasionally live attenuated vaccines are used at day-of-hatch and some used in ovo at 18 days of embryonation [14].

CAV targets the erythroid and lymphoid progenitor cells in the bone marrow and thymus respectively as the destruction of erythroid progenitors in bone marrow results in severe anaemia and depletion of granulocytes and thrombocytes, and the destruction of precursor T cells results in depletion of mature cytotoxic and helper T cells with consequent effects on susceptibility to, and enhancement of, the pathogenicity of secondary infections agents, and sub-optimal antibody responses [15]. Montgomery et al. [16] have shown that their reovirus strain reduced the weight of bursa of Fabricius and spleen, the total numbers of white blood cells in circulation and caused a follicular atrophy in the bursa of Fabricius. These authors reported on a functional reduction in the T-cell response.

Although the immunopathogenesis of CAV and ARV infections singly is already explained further studies are necessary to elucidate the role of CAV and ARV in the aetiology and pathogenesis of BWD.

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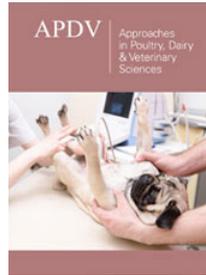
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