Diagnostic Exercise
From The Davis-Thompson Foundation*

Case #: 141 Month: March Year: 2020

Answer Sheet

Title: Circoviral bursitis in nestlings racing pigeons


Figure 1: Bursa of Fabricius, nestling pigeon. Abnormal tissue architecture with lymphoid depletion, randomly distributed cysts formation, and interstitial fibrosis. Cell debris are is noted in the bursal lumen. H&E.

Microscopic Description: The bursa of Fabricius shows abnormal tissue architecture, including several follicles with severe lymphoid cell necrosis and depletion. Atrophic bursal follicles are mainly replaced by numerous, irregular to roughly circular, randomly distributed cysts, most of
which contain fibrinoheterophilic exudate and cell debris. Prominent interfollicular fibrosis is seen.

**Morphologic Diagnosis:** Bursitis, chronic, necrotizing, diffuse, severe, with marked lymphoid depletion, cysts and fibrosis.

**Etiology:** Pigeon circovirus (PiCV).

**Etiologic diagnosis:** Circoviral bursitis.

![Image of histological section](image)

**Figure 2:** Higher magnification of Fig. 1. The lumen of several cysts that replace bursal follicles is occupied by fibrinoheterophilic exudate and cell debris. The interfollicular space is expanded by fibrosis and inflammatory infiltrate. H&E.

**Typical gross/microscopic findings:** At postmortem examination, the atrophy of the bursa of Fabricius is the most common finding caused by the Pigeon circovirus (PiCV) infection in pigeons. Atrophy of the thymus, spleen, cecal tonsils, and gut- and bronchus-associated lymphoid tissue are also consequence of this viral infection. A wide range of other gross lesions affecting the respiratory or and gastrointestinal tracts
associated with immunosuppression and a plethora of secondary bacterial, viral, fungal or parasitic infections may be present.

Microscopically, PiCV infection is characterized by diffuse cortical and medullary lymphoid depletion in the bursa of Fabricius. Atrophic lymphoid follicles are gradually replaced by cystic formations containing cell debris, basophilic amorphous material and/or fibrinoheterophilic exudate with or without bacteria. Intracytoplasmic, and rarely intranuclear, botryoid, basophilic inclusion bodies within macrophages of affected bursal follicles are usually observed. In chronic PiCV-infection cases, inclusion bodies may not be present in affected bursas of Fabricius.

**Discussion:** PiCV, which belongs to the Circovirus genus and Circoviridae family, is considered one of the major pathogens affecting racing, fancy and meat pigeons under one year of age. PiCV infection has been described in the US, Canada, several European countries, Australia and South Africa. To the best of our knowledge, until now this condition has not been reported in Latin America.

PiCV infection, which is characterized by high morbidity and low mortality, typically causes poor racing performance, reduced weight gain, lethargy, anorexia, and a wide range of clinical signs associated with secondary/concurrent infections. PiCV affects the bursa of Fabricius and other lymphoid tissues (thymus, spleen, cecal tonsils, and gut/bronchus-associated lymphoid tissue) of domestic pigeons, producing severe lymphoid depletion and, consequently, immunosuppression. Therefore, PiCV plays a key role in predisposing the infected bird to a plethora of secondary infections including the multifactorial disease widely described in several European countries named 'young pigeon disease syndrome' (YPDS). Also, multiple other diseases such as salmonellosis, colibacillosis, aspergillosis, mycoplasmosis, trichomoniasis, and infections by pigeon poxvirus, pigeon paramyxovirus-1, pigeon adenovirus-1, and pigeon herpesvirus (Columbid alphaherpesvirus-1) infections can also be found in young pigeons infected with PiCV.

PiCV infection is horizontally transmitted by chronic healthy asymptomatic carriers. Both oral and respiratory routes are possible portal of entries in susceptible birds under one year of age. The unusual earlier age of presentation of the disease in the current case strongly suggested the possible occurrence of vertical transmission. This possibility was proposed in few previously reported publications due to evidence of PiCV detection in embryos and 1-day-old pigeons.
In this case, despite the lack of observation of intracytoplasmic, basophilic inclusion bodies compatible with those produced by PiCV in the bursa of Fabricius,1,2,3,5,8,9,10,13 other bursal histopathologic findings compatible with this viral infection together with the absence of vertically transmitted bacterial infections (e.g. Salmonella Typhimurium) suggests that PiCV infection may have been the cause of death of both nestling racing pigeons.

References and Recommended literature:


*The Diagnostic Exercises are an initiative of the Latin Comparative Pathology Group (LCPG), the Latin American subdivision of The Davis-Thompson Foundation. These exercises are contributed by members and non-members from any country of residence. Consider submitting an exercise! A final document containing this material with answers and a brief discussion will be posted on the CL Davis website (http://www.cldavis.org/diagnostic_exercises.html).

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