Title: Chicken proventricular necrosis and lymphocytic proventriculitis.

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Fig. 1. Proventriculus. Moderate infiltration of lymphocytes and fewer macrophages in the interstitial space. HE. 40X.
**Fig. 2.** Proventriculus. Prominent necrosis and sloughing of the epithelial glandular cells into the glandular lumen (white asterisk). There are several lymphocytes and a few macrophages in the interstitial space. Columnar epithelial cells are replacing the normal glandular cells in the proventricular glands (black asterisk). HE. 40X.
**Fig. 3.** Proventriculus. Strong cytoplasmic immunoreaction of the necrotic cells within the glandular lumen and rare nuclear and cytoplasmic immunolabeling of the glandular epithelial cells. Chicken proventricular necrosis virus immunohistochemistry. DAB/hematoxylin counterstain. 40X.

**Diagnosis:** Proventricular glandular degeneration and epithelial necrosis, multifocal lymphocytic proventriculitis, and hyperplastic ductal epithelium.

**Cause:** Chicken proventricular necrosis virus (CPNV) formerly known as transmissible viral proventriculitis (TVP) virus, Birnavirus.

**Typical Gross Findings:**

- Severe and diffuse thickening of the proventricular wall with prominent appearance of the glandular lobules.
- Circular areas of congestion and hemorrhage within the proventricular mucosa.

**Typical Microscopic Findings:**

- Variable amounts of aggregates of necrotic cells, predominantly the oxynticopeptic cells, within the lumen of the proventricular glands.
- Severe proventricular interstitial lymphocytic infiltration.
- Hyperplastic ductal epithelium replacing the glandular epithelium (metaplasia).

**Discussion:** Chicken proventricular necrosis virus (CPNV) causes transmissible viral proventriculitis (TVP), a disease of broiler chickens and commercial laying hens with high economic impact in the poultry industry (Dormitorio et al. 2007, Noiva et al. 2015). The etiological agent was a mystery for several years, with many studies suggesting adenovirus, reovirus, infectious bronchitis virus, infectious bursal disease virus, and picornavirus as possible etiologies (Grau-Roma et al. 2017). However, in 2011, a new birnavirus was detected in natural cases and experimentally able to cause the disease in chickens. This new birnavirus was coined as CPNV (Guy et al. 2011a and b). Since its discovery in 2011, the virus has been associated with other conditions such as runting and stunting syndrome (Noiva et al. 2015).

Flocks affected by CPNV are mostly chickens between four to five weeks of age; however, the disease has been also described in birds between 9 to 20 weeks of age (Marusak et al. 2012). Grossly, CPNV-infected chickens have severe and diffuse thickening of the proventricular wall and prominent proventricular glandular lobules (Grau-Roma et al. 2017). Microscopically, the most important lesions are restricted to the proventriculus, where necrosis of oxynticopeptic cells, lymphocytic infiltration, and hyperplastic ductal epithelium that replaces the glandular epithelium are commonly observed (Goodwin et al. 1996, Hafner and Guy 2013). All these features were observed to varying degrees in the eight chickens submitted for necropsy in this case. The fact that the ventriculus was thinned is not a common finding in CPNV. Microscopically, there was severe heterophilic ventriculitis with bacterial coating. We believe that the thinning of the ventricular muscles may have been caused by the lack of peristaltic movement as a downstream consequence of the proventriculitis, predisposing to the bacterial infection and heterophilic inflammation in the ventriculus. Immunohistochemistry for CPNV was negative in the ventriculus.

**References:**

5. Guy JS, West MA, Fuller FJ, Marusak RA, Shivaprasad HL, Davis JL, Fletcher OJ. 2011b. Detection of chicken proventricular necrosis virus (R11/3 virus) in experimental and naturally occurring cases of transmissible viral proventriculitis with the use of a reverse transcriptase-PCR procedure. Avian Dis. 55:70-75

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