Dilated Cardiomyopathy in a Rio Grande Wild Turkey (*Meleagris gallopavo intermedia*) in Southern Utah, USA, 2013

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**ABSTRACT:** A male Rio Grande Wild Turkey (*Meleagris gallopavo intermedia*) living in semidomestication was submitted for necropsy. Emaciation, a greatly enlarged heart, and chronic passive congestion of the liver were present. Dilated cardiomyopathy occurs in domestic turkey flocks but has not been reported in Wild Turkeys.

Dilated cardiomyopathy (DCM), formerly known as spontaneous cardiomyopathy of turkeys, occurs frequently in commercial domestic turkeys (*Meleagris gallopavo*). An enlarged heart in commercial turkeys was first described in 1962 (Magwood and Bray 1962) and subsequently in 1968 (Sautter et al. 1968). Incidence may be higher in flocks raised at moderate to high elevations (Frame et al. 2001). Reducing the sodium and increasing the chloride concentration in the diet significantly decreased mortality attributable to DCM in pouls up to 3 wk of age (Frame et al. 2001). Simply reducing the salt concentration in the starter diet accomplished similar results (Clark et al. 1995). Rapid growth rate is a factor in occurrence of DCM, as well as the interaction of multiple determinants and risk factors (Frame 1991; Frame et al. 2010). The majority of mortality in affected commercial flocks occurs within the first 3 wk of life when the rate of growth is high. However, DCM has also been seen frequently in tom (male) flocks at 6–8 wk of age, and even sporadically in individual toms as old as 18 wk (D.D.F. pers. obs.). Wild Turkeys (also *M. gallopavo*) do not experience dramatically accelerated growth rates.

Genes from the turkey have been studied as models to better understand human dilated cardiomyopathies (Lin et al. 2007). In Wild Turkeys, a possible DCM-sparing variant of the inhibitory subunit troponin I (Tnl) of the troponin-tropomyosin system that governs skeletal muscle contraction in vertebrates has been reported (Biesiadecki et al. 2004). It is possible that the compensatory variant of the Tnl subunit of the filament-based troponin-tropomyosin system may selectively spare the Wild Turkey population from an appreciable incidence of DCM. In at least three Wild turkey disease surveys from various areas of the US, cardiac abnormalities are not mentioned (Trainer et al. 1968; Davidson et al. 1985; Peterson et al. 2002).

We describe an unusual occurrence of DCM in a mature male Rio Grande Wild Turkey (*Meleagris gallopavo intermedia*) found dead in March 2013 by Utah Division of Wildlife Resources (DWR) personnel in southern Utah (37°28′46″N, 113°18′35″W). To our knowledge, this is the first report describing DCM in a Wild Turkey. The turkey was submitted for necropsy to the Utah Veterinary Diagnostic Laboratory-Central Utah Branch (UVDL-CUB) with a history of sudden death. The turkey had been living in semidomestication on the premises of a recently retired DWR employee. It was submitted to UVDL-CUB with concern that the turkey may have died from infectious contagious causes that might endanger the free-living turkey flock nearby.

The turkey was severely emaciated. Upon necropsy, the peritoneal cavity contained an abundance of yellow serous fluid. Lungs were fluid filled and contained areas of dark red to black consolidation. The spleen was moderately enlarged, and the capsule appeared thickened. There was
widespread congestion of intestinal vasculature. The most striking lesions were a greatly enlarged heart and a firm liver with markedly rounded edges. Right and left ventricular dilatation were present; however, measurements were not taken (Fig. 1). Culture of heart sac, liver, and spleen yielded no bacterial growth.

No microscopic lesions were detected in the myocardium; however, histologic lesions in the liver were supportive of chronic passive congestion with moderate to severe bridging fibrosis, especially prominent in the centrilobular areas. Moderate to marked vacuolation (mostly lipid type) of hepatocytes was also present. Diffuse pulmonary congestion with presumptive mild pulmonary edema, as well as prominent congestion in the kidneys and intestinal tract was observed.

Living in semidomestication may have allowed this turkey to continue surviving with a severely compromised heart. A turkey in this condition would probably not survive in the wild and, consequently, would not be detected on surveys. Any case of sudden death in Wild Turkeys must be taken seriously because of the risk of harboring and transmitting serious diseases, such as avian influenza or exotic Newcastle disease, into wild or domestic turkey populations.

The cause of DCM in this Wild Turkey is unknown. The diet of this semidomesticated bird was probably different than that of wild flocks and may have been a contributing factor in the pathogenesis.

**LITERATURE CITED**


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