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Proventricular Impaction Associated with Nonsuppurative Encephalomyelitis and Ganglioneuritis in Two Canada Geese

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ABSTRACT: Two wild Canada geese (*Branta canadensis*) in an extremely emaciated state and with severe proventricular food impaction also had a nonsuppurative encephalomyelitis and ganglioneuritis. The condition in these two birds was morphologically similar to psittacine proventricular dilatation, a recently identified disease of psittacine birds.

Key words: Canada goose, *Branta canadensis*, proventricular impaction, emaciation, nonsuppurative encephalomyelitis, ganglioneuritis, case report.

Proventricular food impaction combined with severe emaciation is a typical morphologic manifestation of chronic lead poisoning secondary to ingestion of spent lead shot in waterfowl (Friend, 1987; Wobeser, 1981). We describe a condition in two free-flying Canada geese (*Branta canadensis*) characterized grossly by these findings but associated instead with a nonsuppurative encephalomyelitis and ganglioneuritis. Both birds were submitted for necropsy to the Diagnostic Laboratory of the Atlantic Veterinary College, University of Prince Edward Island, Charlottetown, Prince Edward Island, Canada C1A 4P3. One bird (A) was received in June 1988, the other (B) in April 1989. The age and sex of bird A were not determined. Bird B was an adult male. Tissues from each bird were fixed in 10% formalin, processed routinely for light microscopic examination, and stained with hematoxylin and eosin (Luna, 1968). Fresh portions of brain and spinal cord from bird B were kept at −70 C until processed for virological examination. A 30 to 50% homogenate of nervous tissue from this bird in minimum essential medium (MEM) with Earle's salts (Sigma Chemical Company, St. Louis, Missouri 63178, USA) and 500 units/ml penicillin, 500 μg/ml streptomycin, and 0.125 μg/ml fungizone (Flow Laboratories, Mississauga, Ontario, Canada L5S 1R2) was prepared. This homogenate was clarified by centrifugation at 500 × *g* for 15 min. The supernatant was filtered through 0.22 *μm* filter units (Millipore Products Division, Bedford, Massachusetts 01730, USA) and inoculated into 5-day-old Swiss white mice by intracerebral route, 11-day-old chicken embryos by intra-allantoic route, 24-hr-old specific-pathogen-free chicken embryo fibroblasts (SPAFAS, Norwick, Connecticut 06360 USA), and Vero cells (ATCC, Rockville, Maryland 20852, USA). Chicken embryos and suckling mice were monitored for mortality or abnormal behavior, while cell cultures were observed for the presence of cytopathic effects. Passages of inoculated tissues were done three to four times in each case. Smears of nervous tissue from bird B were also examined for the presence of alphaviruses and flaviviruses by the fluorescent antibody technique, using immune grouping ascitic fluids supplied by the National Institutes of Health (Bethesda, Maryland 20705, USA). Portions of kidneys from both birds and of liver from bird B were analyzed for the presence of lead. Ten ml of concentrated ultrapure nitric acid were added to duplicate 1-g portions of fresh kidney and duplicate 0.5-g portions of freeze-dried liver weighed to the nearest 0.01 g. These samples were left overnight at room temperature. They were then digested on a hot plate at medium heat until near dryness and diluted to 10.0 ml with deionized water. Lead analysis of each

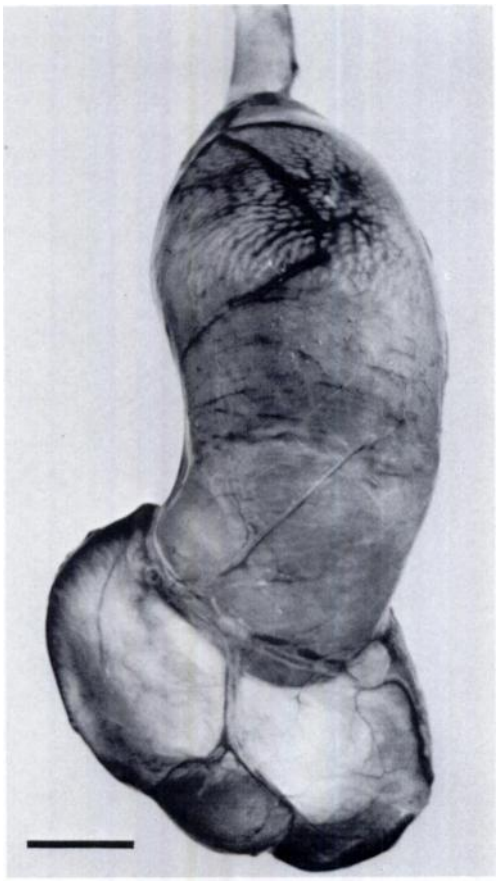

FIGURE 1. Marked proventricular dilatation associated with food impaction in a Canada goose with nonsuppurative encephalomyelitis and ganglioneuritis. The esophagus (top) and gizzard are not affected. Bar = 2 cm.

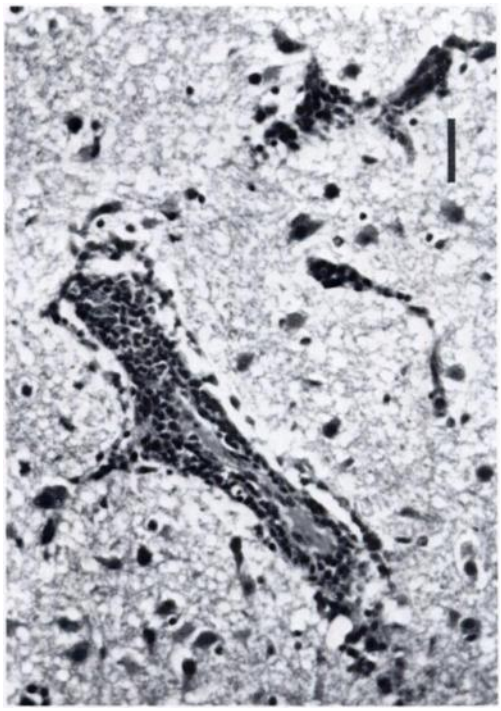

FIGURE 2. Portion of brain stem from a Canada goose with nonsuppurative encephalomyelitis and ganglioneuritis. The blood vessels are cuffed by numerous lymphocytes and plasma cells. H&E. Bar = 30 μm.

sample was done by flame atomic absorption spectroscopy (Perkin-Elmer Corporation, Ridgefield, Connecticut 06877, USA). International Atomic Energy Agency (A-1400 Vienna, Austria) certified reference material (MA-A-ITM–copepod) was used as a positive control.

The salient gross lesions in both birds were extreme emaciation, characterized by almost complete atrophy of the pectoral muscles and loss of internal fat reserves, and marked proventricular dilatation (approximately five times normal size) associated with food impaction but with an intact proventricular mucosa (Fig. 1). The impacted food consisted of partly digested grass which, in bird B, was mixed with some grain. In bird B, the gizzard also appeared moderately distended by bile-stained ingesta, and the distal region of the large intestine over a length of about 7 cm had a necrotic mucosal surface and was distended by foul-smelling material that included urate and fibrin.

In both cases, the most prominent microscopic lesion was a nonsuppurative encephalitis characterized primarily by perivascular cuffing by lymphocytes and plasma cells (Fig. 2). The nervous parenchyma itself showed very mild changes that included a few small microglial nodules and neuronophagia of a very few necrotic neurons. In bird A, lesions were most prominent in the medullary region of the brain stem but also occurred in the cerebrum and cerebellum. In bird B, lesions were of comparable degree in the brain stem and cerebrum but were very mild in

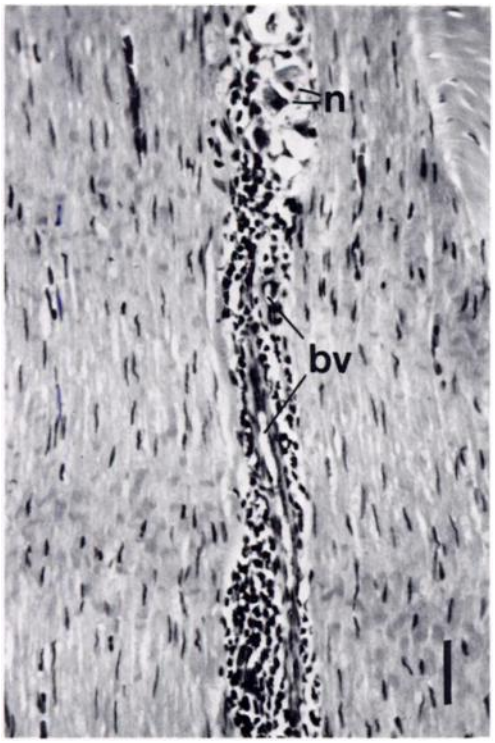

FIGURE 3. Small ganglion of myenteric plexus in the gizzard of a Canada goose with nonsuppurative encephalomyelitis and ganglioneuritis. The neuronal cell bodies (n) are intact, but adjacent small blood vessels (bv) are cuffed by numerous mononuclear leukocytes. H&E. Bar = 30 μm.

the cerebellum. The spinal cord of both birds had lesions similar to those in the brain and involving primarily the gray matter. Lesions in all other tissues were vary mild. In bird A, many areas of the submucosal and myenteric plexuses and their associated autonomic ganglia along the esophagus, proventriculus and gizzard were infiltrated by a small to moderate number of mononuclear leukocytes. One small epicardial autonomic ganglion was similarly affected. In many of these areas, the inflammatory cells were in close proximity to, or surrounding, small blood vessels and were therefore interpreted as components of perivascular cuffs (Fig. 3). There was no evidence of neuronal necrosis in affected ganglia. In bird B, infiltration of a few areas of the submucosal and myenteric plexuses by a very small number of mononuclear leukocytes was seen only in the proventriculus and in the distended distal region of the large intestine. Many mucosal glands in the proventriculus of this bird contained trematodes tentatively identified as *Tetrameres* sp., and the distended portion of the large intestine showed evidence of mucosal necrosis with secondary inflammation and fibrosis. Therefore, the possibility that inflammatory changes in the submucosal and myenteric plexuses of the proventriculus and large intestine of this bird were part of a local inflammatory reaction rather than part of a generalized ganglioneuritis could not be ruled out. The vagus nerves of this bird contained a very few small aggregates of lymphocytes and plasma cells, some of which were around small blood vessels. All attempts at virus isolation from the central nervous system of bird B were unsuccessful. In both birds, the kidneys contained less than 1 μg of lead per gram of wet tissue. According to Wobeser (1981), lead levels in kidneys of waterfowl poisoned by lead are consistently above 6 μg per gram of wet tissue. The liver of bird B contained less than 2 μg of lead per gram of dry tissue, as compared to levels of 20 to 30 μg/g on a dry-weight basis in cases of lead poisoning in waterfowl (Friend, 1987).

Causes of esophageal and/or proventricular impaction can be mechanical or neurogenic. Crop impaction may occur as a result of overeating in young male budgerigars or because of overly fibrous food or hair mixed with food in insectivorous birds (Minsky and Petrak, 1982). Impaction of the crop and esophagus with long tightly interwoven blades of coarse grass may occur in domestic poultry if the birds are kept off feed for some time prior to being put on range (Peckham, 1978). In a die-off of about 2,000 Canada geese at Lac Qui Parle State Wildlife Refuge in Minnesota (USA) in 1986, the main lesion in affected birds was an impaction of the esophagus that ended at the thoracic inlet. This impaction was accompanied by necrosis and ulceration of the esophageal mu-

cosa and was associated with mechanical injury to the esophageal wall by fox tail grass (*Setaria lutescens*) seeds (K. Roertgen and R. Windingstad, pers. comm.).

Lead poisoning in waterfowl often causes impaction of the proventriculus and lower esophagus with food (Friend, 1987; Wobeser, 1981). This is thought to result from a peripheral neuropathy which interferes with the normal peristaltic movements of the digestive tract. In mallard ducks experimentally poisoned with lead, the vagus nerves were consistently more severely affected than peripheral nerves supplying skeletal muscles (brachial, sciatic) (Hunter and Wobeser, 1980). The vagus nerves are the principal motor nerves to the esophagus, crop, proventriculus and gizzard (Sturkie, 1970).

A recently identified disease of psittacine birds, known variably as psittacine proventricular dilatation, macaw wasting syndrome, myenteric ganglioneuritis, and neuropathic gastric dilatation of psittaciformes, shows the closest morphologic similarity to the cases described herein, since it combines the unique features of proventricular food impaction and nonsuppurative encephalomyelitis and ganglioneuritis (Hughes, 1984). This condition is almost invariably fatal and is characterized morphologically by emaciation, distention of the esophagus and proventriculus with food, multifocal lymphocytic encephalitis (present most consistently in the brain stem) and poliomyelitis, and multifocal leiomyositis of the proventriculus and gizzard accompanied by depletion of neurons in, and inflammation of, the myenteric plexus. The cause of this condition is unknown. The nervous lesions are suggestive of a viral cause. However, all attempts at viral isolation have so far been negative, although scattered virus-like particles have been seen ultrastructurally in affected nervous tissue (Gerlach, 1986). The disease does not appear to be highly contagious and may have a long incubation period (Woerpel and Rosskopf, 1984; Phalen, 1986).

Conditions in mammals bearing some clinical similarities to those in psittacine birds and in the Canada geese in this report are feline dysautonomia and equine grass sickness. The causes of these two conditions are presently unknown. Both diseases are characterized morphologically by degenerative changes in neurons of autonomic ganglia of the sympathetic and parasympathetic nervous systems, dorsal root ganglia, ganglia of cranial nerves, and ventral horns and intermediolateral gray matter of the spinal cord. However, an inflammatory response is not a prominent feature of either disease (Sharp et al., 1984).

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