CROSS-CANADA DISEASE REPORT

RAPPORT DES MALADIES DIAGNOSTIQUÉES AU CANADA

Eastern Canada

Eastern Canada rabies diagnoses, January 1 – June 30, 1992

From January 1 to June 30, 1992, 5257 specimens were tested for rabies at the Animal Diseases Research Institute (ADRI), Nepean. Of these, 1141 (21.7%) were found to be positive. The rabies cases from Quebec are part of an ongoing epizootic that began in 1989 when rabies spread from Ontario across the Ottawa River near Pembroke, Ontario. From the Outaouais region, the spread has been northward to the Mont Laurier/Grand Remous region, and eastward to the Joliette region north of Montreal. At about the same time, rabies reentered Quebec from New York State near Huntingdon, Quebec, and has spread northward to the St. Lawrence River and eastward to the St. Hyacinthe area.

In Ontario, in recent years, rabies spread northward along Georgian Bay and then westward toward Sault Ste. Marie. In 1992, this epizootic continued to spread northward into the Kirkland Lake area of Ontario and into the Rouyn-Noranda region of Quebec. In 1989, in two large areas of eastern Ontario, the Ontario Ministry of Natural Resources conducted an oral vaccination campaign, using bait containing the Evelyn, Rokitniki, Abelseth (ERA) rabies vaccine. This campaign was repeated in 1990, and again in 1991, to cover all areas of Ontario east of a line between Pembroke and Kingston and selected areas of southwestern Ontario. **Nearly two million packages of bait were dropped from airplanes or distributed by hand during these years. The immunization campaigns have markedly reduced the number of samples from eastern Ontario that have been diagnosed positive.**

The foxes from Newfoundland were from the mainland of Labrador. There have not been any documented cases of rabies on the island of Newfoundland since 1989.

All rabid specimens have arctic and subarctic zones and from the fox vaccination area of Ontario, all rabid bats, raccoons, coyotes, and wolves, and all geographically isolated and unusual cases of rabies are subjected to virus typing by monoclonal antibodies. One hundred and three monoclonal antibodies produced at ADRI and at the Swiss Rabies Centre are used for this purpose. **Monoclonal antibodies allow differentiation among the arctic strain (in carnivores in northern zones and in foxes and skunks in Ontario and Quebec), several different strains occurring in bats, the raccoon virus presently spreading northward in New York State, the ERA vaccine virus used for oral immunization of foxes, and numerous exotic strains and rabies-like viruses.** The viruses from 559 rabid specimens received during this period were typed. A rabid skunk received on February 21, 1992, from southwestern Ontario yielded a "Big Brown Bat Type I" virus. No other unusual finding was made.

Table 1: Specimens positive for rabies by species and province/territory in eastern Canada from January 1 to June 30, 1992

Animal	Quebec	Ontario	Newfoundland Labrador	NWT	Other	Totals
	*	*	*	*	*	*/**
Cattle	34	82				116/484
Cat	19	27				46/992
Dog	42	26		1		69/987
Goat	5	1				6/35
Horse	3	7				10/55
Pig		11				11/20
Sheep	4	9				13/74
Bat	2	5				7/142
Black Bear		2				2/9
Coyote	3	7				10/68
Fisher		1				1/1
Fox	319	352	10	1		682/932
Raccoon	15	10				25/578
Skunk	34	104				138/302
Wolf	5					5/18
Other						0/560
Totals	485/2008	644/3183	10/28	2/4	0/34	1141/5257

* = positive/** = examined

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Ontario

Lungworm (*Crenosoma vulpis*) infection in dogs

In October, 1992, a diagnosis of *Crenosoma vulpis* infection was made in four dogs from the central part of Ontario. The diagnosis was made from transtracheal washes and sputum smears from coughing dogs, and identification of the parasite was confirmed by fecal flotation and water Baermann procedures.

All dogs appeared bright and alert on clinical examination. A dry, retching cough could be elicited by gentle tracheal palpation in all dogs. The dogs were living in rural areas, and were free to roam at times. Red foxes were known to live in the area, and an increase in gastropods (slugs and snails) had been noted by one

owner, possibly associated with the wet summer and fall.

Crenosoma vulpis is a metastrongyloid parasite found in the bronchi of canids. Infection is common in foxes and possibly other canids throughout the world (1). Very few reports in dogs are available in North America (2). Animals are infected by eating a gastropod containing third-stage larvae. Once ingested, the larvae migrate from the small intestine, through the liver to the lungs. About 17 days after infection, adult lungworms are present in the bronchi and may be found in tracheal mucus and feces (1).

Two dogs were treated with levamisole and the other two were treated with ivermectin. Clinical signs began to resolve during the first week of treatment, and the dogs were free of signs within two weeks. Larvae were not found in fecal samples examined two and four weeks after completion of treatment.

References

1. Cobb MA, Fisher MA. *Crenosoma vulpis* infection in a dog. Vet Rec 1992; 130: 452.
2. Stockdale PHG, Hulland TJ. The pathogenesis, route of migration, and development of *Crenosoma vulpis* in a dog. Pathol Vet 1970; 7: 28–42.

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Alberta

Strychnine toxicity in livestock

Strychnine is frequently identified as the agent of malicious poisoning in companion animals, but is generally not considered in the differential diagnosis of acute death in livestock. Strychnine is an indole alkaloid that acts by direct, selective antagonism of spinal inhibition. Elimination of reflex control results in extensor rigidity and convulsions (1). Clinical signs include apprehension and stiffness that progress rapidly to rigidity and tetanic seizures, but increase in intensity and duration until death from anoxia occurs. Strychnine is governed by legislation restricting its use to pest control agents and farmers, principally for the control of burrowing rodents, skunks, and coyotes. Accidental poisoning occurs when non-target species consume either grain baits or the bodies of poisoned animals, which may contain sufficient quantities of bait in the stomach to be lethal.

There are no diagnostic postmortem lesions to implicate strychnine toxicosis, and gross or microscopic lesions are not present in the central nervous system, so diagnosis is confirmed by the detection of the compound, generally in stomach contents or liver (1). An index of suspicion to request analysis must exist, and is generally based on a history of exposure. Sudden death, rapid onset and decline of rigor, pulmonary congestion, and the presence of a large quantity of stomach contents support the suspicion. Between April and July 1992, strychnine toxicity in a sow and two horses was confirmed by staff of the Animal Health Laboratories Branch, Alberta Agriculture. In all cases, there was a history of potential contact, without which the diagnosis would not have been made.

The sow originated from a herd of 325 animals in a farrow-to-finish operation in which 10 sows had died in four months. One animal had been examined and was found to have an intestinal torsion. This sow was found gasping for breath and foaming at the mouth; she was hosed down to cool her off, but she died within three hours. The submitter mentioned that poison to kill ground squirrels has been placed in the barn to control mice, but the sows had no contact with it. The sow was very autolyzed and had severe pulmonary edema and congestive atelectasis, with a large amount of bile-stained liquid and a small amount of grain in the stomach. Analysis of the stomach contents detected 7.1 mg% strychnine.

Two horses, a mare and a gelding, from a herd of 10, were found dead after having been observed to be normal in the afternoon of the day before. The mare was found on the outside of a Texas gate, and it was assumed that she had jumped it. One cow had died the day before, and when questioned about potential exposure to toxins, the owners mentioned that bait for ground squirrels has been placed in the pasture two months previously, but that it was all located in burrows. The mare had multiple fractures of the pubis and acetabulum, a diaphragmatic hernia and severe hemothorax, with hemorrhage extending into the abdomen through the hernia. Stomach contents consisted of a scant amount of grain. Strychnine content was 3.9 mg%. The gelding was autolyzed and had severe pulmonary congestion; its stomach was distended with a large quantity of chopped grain and a few whole kernels. Strychnine levels were 21.4 mg% in the stomach contents. It is probable that the gelding consumed enough strychnine to make it rapidly lethal, whereas the mare consumed sufficient drug to result in convulsions, possible entrapment in the gate, and subsequent trauma. The cow was severely autolyzed, and analysis of rumen contents was negative, but dilution of potent toxins by the volume of the rumen contents may prevent detection.

Without the history of potential exposure to rodenticide in these cases, the correct diagnosis would not have been made. Sudden, unexplained death in livestock species is recognized in all diagnostic laboratories and veterinary practices. These cases illustrate the critical aspects of history in diagnosis, and serve as a warning that strychnine toxicity may be claiming more livestock than we suspect.

Reference

1. Osweiler GD, Carson TL, Buck WB, VanGelder GA. Clinical and Diagnostic Veterinary Toxicology. 3rd ed. Dubuque, Iowa: Kendall/Hunt Publishing Company, 1985: 345–348.

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