ARTICLES

Paramyxovirus-1 in feral pigeons (*Columba livia*) in Ontario

Kathleen M. Johnston, Douglas W. Key

Abstract

Paramyxovirus-1 (PMV-1) infection was diagnosed in racing pigeons in Ontario during 1985, but it was not until January 1989, that the virus was isolated from feral pigeons (*Columba livia*) in this province. During an 18 month period beginning January 1988, a total of 43 feral pigeons was submitted to the Wildlife Diseases Laboratory, Pathology Department, Ontario Veterinary College. A history of neurological signs accompanied most of the birds. Tissues from 29 birds were submitted for PMV-1 isolation. Allantoic inoculation of embryonated chicken eggs yielded PMV-1 in 10 of the pigeons submitted. On the basis of histological criteria, we believe that 12 other birds were also infected with PMV-1.

Gross pathological changes were unremarkable. Lymphplasmacytic interstitial nephritis was observed histologically in all birds from which PMV-1 was isolated. Other lesions seen, in decreasing frequency of occurrence, were lymphoplasmacytic interstitial hepatitis and multifocal hepatic necrosis, lymphoplasmacytic interstitial pancreatitis, nonsuppurative encephalitis and myelitis.

The existence of PMV-1 in feral pigeons poses a potential threat to the poultry population since there is ample opportunity for mingling with poultry under open housing management. There is also a concern that pigeons may harbor the virus, perhaps in the kidney, and become chronic carriers and potential long-term disseminators of the disease.

Résumé

Virus paramyxo-1 chez les pigeons sauvages (*Columbia livia*) en Ontario

L'infection virale causée par paramyxo-1 (VPM-1) a été diagnostiquée chez les pigeons voyageurs en Ontario, en 1985. Toutefois, ce n'est qu'en janvier 1989 que le virus a été isolé chez les pigeons sauvages (*Columbia livia*) de cette province. Quarante-trois pigeons ont été soumis, sur une période de 18 mois depuis janvier 1988, au Wildlife Diseases Laboratory, Pathology Department, Ontario Veterinary College. L'anamnèse indique que des signes neurologiques étaient présents dans la plupart des cas. Des échantillons tissulaires provenant de 29 oiseaux ont été soumis pour isolement du VPM-1. Le test d'inoculation sur embryon de poulet a permis de déceler la présence du VPM-1 chez 10 pigeons. Selon les critères histologiques, les auteurs concluent que 12 autres pigeons étaient aussi infectés par le VPM-1. Les changements macroscopiques étaient peu remarquables. Une néphrite interstitielle lymphoplasmocytaire a été observée chez les sujets dont l'inoculation a donné un résultat positif. Les autres lésions observées par ordre décroissant furent : une hépatite interstitielle lymphoplasmocytaire et nécrose multifocale, une pancréatite interstitielle lymphoplasmocytaire, une encéphalite non suppurée et une myélite. La présence du VPM-1 chez les pigeons sauvages pose un danger potentiel à la population aviaire puisqu'il y a beaucoup d'occasion de contact physique avec les élevages de volailles gardées dans des fermes à aires ouvertes. Les auteurs craignent aussi que le pigeon soit porteur du virus, au niveau des reins et qu'il puisse ainsi disséminer la maladie sur une période de temps indéterminée. *(Traduit par Dr Thérèse Lanthier)*

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Introduction

In 1971-73, an epornitic of Newcastle disease (ND) in poultry in Europe was associated with a similar outbreak in racing pigeons (*Columba livia*), caused by a velogenic strain of paramyxovirus-1 (PMV-1) indistinguishable from the epornitic virus in poultry (1). It was assumed that the pigeons were infected by diseased domestic poultry. In 1981, a disease resembling neurotropic ND was seen in racing pigeons in the Mediterranean. Subsequently, the virus was isolated from two diseased racing pigeons imported into

Department of Pathology, Ontario Veterinary College, University of Guelph, Guelph, Ontario N1G 2W1 (Johnston), and Ontario Ministry of Agriculture and Food, Veterinary Services Laboratory, P.O. Box 3612, Guelph, Ontario N1H 6R8 (Key). Present address of Dr. K.M. Johnston: Department of Veterinary Pathology, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, Saskatchewan S7N 0W0.

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Table 1. Summary of clinical signs, results of virus isolation and histological lesions observed in feral pigeons in Ontario diagnosed as having PMV-1 infection

Case number	Clinical signs[a]	Results of virus isolation by organ[b]									Histological lesions[c]					Diagnosis of PMV-1	
		B	H	L	R	K	S	P	T	C	K	L	P	B	M	Virology[d]	Histology[e]
1	N	−	f	−	−						+	+		−	−	−	+[g]
2	N	−		−		−				−	+	+	−	−		−	+[g]
3	D	+	+	+	+		+			+	+	−	+	−	−	+	+
4	N	−		−	−	−	−				+	+		+		−	+[g]
5	N	−		−	−	−	−				+	+	−	+		−	+[g]
6	FD	−	−	−	−	−	−			−	+	+	+	+		−	+[g]
7	N	−	−	−	−	−	−			−	+	+	−	−		−	+[g]
8	N	+	−	−	−	−	−	−			+	+	+	+	+	+	+
9	N	−									+	+	+	−		−	+[g]
10	N	+	+	+	+	+				+	+	+	+	+		+	+
11	NH	−	+	+	+	+				−	+	+	−	+		+	+
12	N	+		+	+	+			−							+	
13	N	+		+	+	+			−							+	
14	N						+									+	
15	N										+	+		−			+[g]
16	N										+	+		−			+[g]
17	N	+		+	−	+					+	+	+	−		+	+
18	N	−			−				−		+	+	−			−	+[g]
19	FD	−		−		−	−				+	+	+			−	+[g]
20	NH	−		+		+	+				+	+	−			+	+
21	N,D	−				−	−				+	+				−	+[g]
22	N,D	+		+			+				+	+	+			+	+

[a] N = neurological deficit; D = bright green diarrhea; FD = found dead; NH = no history
[b] B = brain; H = heart; L = liver; R = lung; K = kidney; S = spleen; P = pancreas; T = trachea; C = colon, cloaca
[c] K = kidney; L = liver; P = pancreas; B = brain; M = spinal cord
[d] By allantoic inoculation
[e] + ve diagnosis requires characteristic histological lesions in at least two organs
[f] No sign (+/−) indicates "not examined"
[g] Presumptive diagnosis made on histological lesions

Belgium from Italy (2). During 1983, the virus spread in racing pigeons in Europe as a result of extensive trade and mixing of birds during races (3). In 1983, an epornitic of ND occurred in the unvaccinated, highly susceptible, poultry population of Great Britain, and resulted in the slaughter of 800,000 birds. Epidemiological studies traced the epornitic to the feeding of unprocessed rice bran contaminated with PMV-1 by feces of infected feral pigeons at the Liverpool and Birkenhead docks. Subsequently, the virus was responsible for epornitics of disease in feral pigeons throughout England and Wales. At the same time, there was a dramatic increase in the number of cases of PMV-1 in racing pigeons in the country, likely as a result of interaction between racing and feral pigeons (4).

In 1984, the virus was isolated from pigeons in New York City, which heralded its appearance on the North American continent (5). Its advent in Canada occurred during 1985 in racing pigeons in Ontario (R. J. Hampson, personal communication). Reports of the virus in racing pigeons in Ontario and western Canada followed (6, 7). Until January 1989, the virus had not been recognized in feral pigeons in Ontario. This paper reviews the clinical signs, histology and virus isolation of PMV-1 in feral pigeons submitted to the Wildlife Diseases Laboratory, Ontario Veterinary College, from January 1988 to April 1990.

Materials and methods

Between January 1988 and April 1990, 43 feral pigeons were submitted to the Wildlife Diseases Laboratory of the Ontario Veterinary College (OVC), Guelph, for diagnosis of cause of death. In most cases, routine postmortem examinations were done, tissues were fixed in 10% buffered formalin, processed routinely, and stained with hematoxylin and eosin (H&E) for histological examination. Ancillary procedures, such as bacteriological culture and toxicological analysis, were performed when indicated.

Tissues from 29 pigeons were submitted for viral isolation. Tissues submitted included brain, heart, liver, lung, kidney, spleen, pancreas, trachea, and colon (Table 1). Tissues were ground and resuspended at a dilution of 1:5 in tryptose phosphate broth containing 50 μg/mL of gentamicin. The tissue suspensions were centrifuged at 1000 rpm for five minutes and the supernatant fluids were passed through a 0.45 μm filter to remove bacteria. The filtrates were inoculated in 0.2 mL volumes into the allantoic cavity of nine to eleven-day-old embryonated chicken eggs. After incubation at 37°C for seven days, allantoic fluids were harvested and blind passaged up to three times. Microtiter hemagglutination (HA) tests were conducted on allantoic fluids collected from eggs in which the embryo had died after more than 24 h of incubation, or at the termination of the final passage.

Serial twofold dilutions of the allantoic fluid in phosphate buffered saline and 0.5% chicken erythrocytes were employed. Microtiter hemagglutination inhibition (HI) tests were conducted on HA positive allantoic fluids using four HA units and twofold dilutions of an antiserum to Newcastle disease virus (NDV). The Hitchner B1 strain of NDV was used as a positive control in the test.

Results

Feral pigeons were submitted from sites ranging from Brockville in southeastern Ontario to Owen Sound in southwestern Ontario, and included Toronto, Guelph, Galt, St. Catherines, and Niagara. The largest number of birds came from the centers closest to the OVC. Sixteen submissions were obtained from the campus of the University of Guelph. Pigeons from Toronto were sent from the Toronto Humane Society.

In most cases, pigeons were submitted dead to the OVC, and clinical signs were reported by the person making the submission. Submissions arrived from humane societies and the general public, and all birds were submitted whole. Clinical signs observed are given in Table 1. Neurological deficits prior to death were almost invariably described. These signs included ataxia, head tremor, torticollis, opisthotonos, circling, disorientation, and paralysis of wings or legs. Green diarrhea was described in three birds. Seven birds were found dead, and no history was included for two birds.

Although neurological signs were described in all birds submitted, paramyxovirus infection was not implicated in the deaths of 21 of the 43 pigeons submitted; on this basis, they were excluded from the study. Tissues from nine of these birds were submitted for virus isolation, as they had had signs of neurological disease with no other apparent cause. Paramyxovirus-1 was not isolated from any of these submissions. Neurological signs were attributed to confirmed (n = 5) or suspected (n = 1) 4-aminopyridine toxicity. Diagnoses among the remaining 15 pigeons included diazinon toxicity (n = 2), lead poisoning and gizzard impaction (n = 1), fractures with *Escherichia coli* septicemia (n = 2), degenerative arthropathy (n = 1), subdural hemorrhage (n = 1), and no diagnosis (n = 8).

Tissues for virus isolation were submitted from 20 of the remaining 22 birds (Table 1). The tissues selected were not consistent, because the birds were submitted to the necropsy service at the OVC and the tissues submitted reflected the choices of individual prosectors. Tissues from 10 birds yielded PMV-1. Virus was isolated with approximately equal frequency from the brain, liver, lung, kidney, and spleen. It was recovered with high frequency from the heart, but this organ was submitted only five times. Virus was not isolated from the single pancreatic and the three tracheal samples, and only twice from samples of colon or cloaca.

Gross lesions were not observed in any of the pigeons examined. Histological lesions observed included: nonsuppurative encephalitis and myelitis, extensive infiltrates of lymphocytes and plasma cells in the interstitium of the pancreas, and similar, but less severe, changes in the liver and kidney. These histological lesions are consistent with a diagnosis of paramyxovirus infection (8–12). In all 10 birds from which virus was isolated and on which histological studies were performed, characteristic microscopic lesions were present in tissues from two or more organs (Table 1). On this basis, we made a presumptive, but not absolutely definitive, diagnosis of PMV-1 infection in birds with similar combinations of histological lesions, in the absence of viral isolation, as these combinations of lesions were completely compatible with this diagnosis. Of all birds examined microscopically, those from which PMV-1 was isolated had characteristic renal lesions, and the remainder, from which virus was not isolated, or in which virus isolation was not attempted, had lesions of one or more of kidney, liver, pancreas, or central nervous system. The prevalence of microscopic lesions is summarized in Table 1. In birds from which PMV-1 was isolated, and from others diagnosed on histological grounds as PMV-1, the most common microscopic lesion was lymphoplasmacytic interstitial nephritis. Intertubular infiltrates of lymphocytes, plasma cells, and smaller numbers of heterophils were associated with tubular atrophy (Figure 1). There were random, small foci of hepatic necrosis with inflammatory responses characterized by lymphocytes, plasma cells, and a few heterophils. In the pancreas, lymphocytes and plasma cells were present in the interstitium among the pancreatic acini. Occasionally, large areas of pancreatic parenchyma were replaced by the lymphoplasmacytic infiltrate.

The most prominent change within the central nervous system was the presence of infiltrates of mononuclear cells within Virchow-Robins spaces throughout the brainstem and the cerebellum (Figure 2). Within the contiguous neuropil, there were diffuse infiltrates of inflammatory cells. Rarely, foci of gliosis were evident. Occasionally, there was nonsuppurative meningitis, particularly in perivascular locations. In the single bird whose spinal cord was examined, there was cervical myelitis with focal neuronal necrosis in the ventral horn and associated axonal swelling. Demyelination was not seen.

Pathotyping studies done in chickens on Canadian pigeon isolates of PMV-1 in 1985 versus those done in 1989 have shown a change in values, indicating a shift from the consistent lentogenic pathotype in 1985 to some mesogenic pathotypes in 1989 (P. R. Ide, personal communication). The average of mean death time values of 12 isolates decreased to 88.2 h (range, 73–104) in 1989 from a value of 133 h (range, 107–148) in 1985. Intracerebral pathogenicity index (ICPI) values in day-old chicks increased from 0.63 in 1985 to 0.89 in 1989 and intravenous pathogenicity index (IVPI) values in six-week-old chickens increased from zero in 1985 to a high of 1.3 (range, 0–1.3) in 1989.

Discussion

The high prevalence of neurological signs in these birds indicated that the strain of PMV-1 involved was neurotropic for pigeons, and virus was isolated from seven of 19 brains examined. In poultry, ND virus reaches the choroid plexus and endothelial cells of vessels in the brain within three days following respi-

ratory infection, and viral antigen is present in neurons from days 4 to 8, after which it is no longer detectable within the central nervous system (13, 14). The duration of infection in these pigeons is unknown, but a prolonged course might explain failure to isolate PMV-1 from all brains cultured. Replication of virus within neurons results in neuronal degeneration and gliosis, and an associated perivascular mononuclear cell response, as we observed in 6 of 14 brains examined microscopically. A more detailed neuropathological examination might have revealed more lesions in brain.

Lymphoplasmacytic interstitial nephritis was present in all birds from which the virus was isolated, and indicated a renal tropism in this species. It is not clear whether the kidney is a primary or secondary site of viral replication. Persistence of virus in the kidney, with intermittent cloacal shedding, may be the means of dissemination of the disease in pigeons (15, 16). Histological evidence of pancreatitis was present in all birds from which the virus was isolated. Hepatitis occurred in nine of the ten birds from which virus was isolated.

The absence of respiratory and ocular signs was consistent with the observations of others on PMV-1 infection in pigeons (12). Rapid clearing of the virus may account for the failure to isolate it from the tissues of some of the birds with clinical signs and histological lesions consistent with a diagnosis of PMV-1 infection. In one study, in poultry infected by nasal or conjunctival inoculation, peak NDV titers were reached by day 5, and by day 9 viral antigen was undetectable in all tissues (14).

Infection of feral and racing pigeons in Canada with the ND virus poses a potential threat to domestic fowl. The increase in virulence of the Canadian strain of pigeon PMV-1 is in contrast to the results of comparisons made between isolates of PMV-1 in Great Britain over the period between July 1983 and June 1985 (17). In that study, a decrease in virulence for chickens was shown by a gradual decline in ICPI values over the period of 24 months. Newcastle disease virus isolates and strains have been shown to consist of a cluster of genetically distinctive and mutually tolerant subpopulations. It is possible for a cluster of these subpopulations to be transmitted from host to host with little loss of genetic diversity. In less virulent strains, one or two subpopulations usually dominate the cluster, while with velogenic stains, as many as four to seven distinguishable plaque types are not uncommon. It has been suggested that the transfer of the cluster of subpopulations during an epornitic would increase the chance of survival of the virus in a partially immune host or an alternate host species (18). It has also been shown that the enhancement of virulence of an isolate is not necessarily exclusive to one host species (19). The existence of clusters of subpopulations within strains and the variation between the strains in their ability to enhance virulence in more than one species on serial passage may explain the difference seen between the increasing virulence for chickens of Canadian pigeon isolates of PMV-1 between 1985 and 1989, compared to the decreasing virulence of the British isolates made between 1983 and 1985.

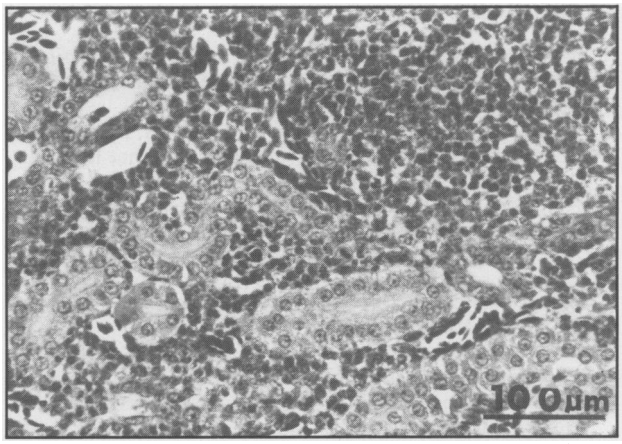

Figure 1. Kidney of a feral pigeon with PMV-1 infection. Multifocal to coalescing infiltrations of lymphocytes, plasma cells, and small numbers of heterophils are present in the interstitium, associated with atrophy of the adjacent tubules. H&E.

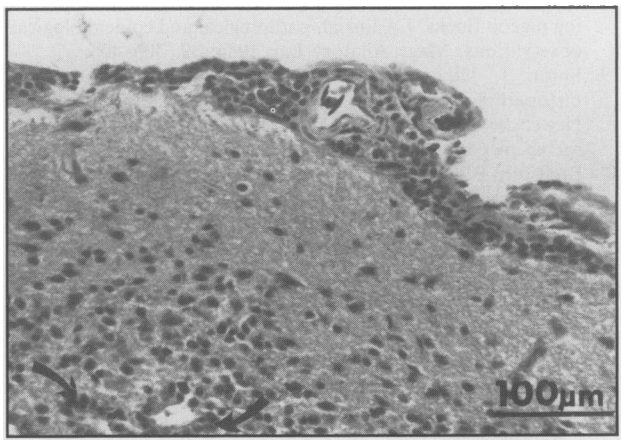

Figure 2. Brain of a feral pigeon with PMV-1 infection. There are mononuclear cell infiltrates around meningeal vessels and within Virchow-Robins spaces (arrows) and surrounding neurophil. H&E.

Unlike the poultry population in Great Britain during the 1984 outbreak of ND, breeders of all commercial poultry types and commercial egg layers in Canada are vaccinated against NDV. However, the majority of commercial chicken broilers and commercial turkeys are not vaccinated and, as a result, are at risk. Passage of the virus through pigeons could potentially result in the emergence of a strain having increased pathogenicity for chickens or other domestic poultry. Awareness of this possibility requires the continued vigilance of provincial and federal government laboratories to monitor the disease in pigeons through viral isolation and pathotyping.

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