# Communication

# Note on the pathology of experimental infection of pigeons by the pigeon paramyxovirosis type I (PPMV-1)

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L'infection expérimentale du pigeon par le paramyxovirus sérotype I (PPMV-1), par voie orale, intramusculaire ou intraveineuse, a occasionné des signes nerveux et de la diarrhée. L'examen *post-mortem* a montré une congestion faible à modérée des viscères et des cas d'entérite catarrhale. Les changements histo-pathologiques étaient plus fréquents dans le système nerveux central, suivi par ceux des reins, du foie, du pancréas, des intestins et des poumons, associés à une méningite encéphalitique bénigne à sévère mais non purulente. *Mots clés* : Pigeon - Paramyxovirus - Infection expérimentale - Soudan.

#### Introduction

Outbreaks of a viral disease in pigeons characterized by a sudden death, nervous signs and great losses in both adults and young birds were reported in 1985 for the first time in the Sudan (4). This virus belongs to the pigeon paramyxovirus subgroup distinguishable from the classical paramyxovirus type I virus, Newcastle disease virus (1, 2). The disease is believed to have spread from East Africa into Europe, especially in Great Britain (10, 11).

The pathological changes were described as enteritis or petechial haemorrhages in the viscera and mononuclear cell infiltration in the brain and visceral organs (11, 12).

This paper reports the gross and histopathological changes in pigeons experimentally infected with the pigeon paramyxovirus type I (PPMV-1).

#### **Materials and methods**

#### Pigeons

Healthy pigeons (2-3 weeks old) of an indigenous breed raised for human consumption and free of PPMV-1 antibodies were used. They were kept under observation for 4-5 weeks (before infection) to guard from inapparent endemic infection. They were provided with water containing copper sulphate (1:2000) for 5 days and metronidazole (Flagyl 400<sup>e</sup>, May and Baker) at the rate of 12.5 mg per livepound suspended in water (0.5 ml per pigeon). Each of them received 0,5 ml terramycin injected intra-muscularly for 3 consecutive days against inapparent bacterial infection.

#### Inoculum

The PPMV-I strain was originally isolated from a natural disease outbreak in pigeons (4). This strain was found to be undistinguishable from the European PPMV-I subgroup which caused great losses in fancy pigeons in continental Europe and Great-Britain (2).

The virus was propagated in 9-day old chicken embryos inoculated via the allantoic cavity to a titre of 10<sup>5</sup> of the virus suspension corresponding to chicken embryo lethal dose 50 (CELD<sup>50</sup>) which was calculated according to REED and MUENCH (14).

#### Experimental design

A total of 38 pigeons were used in 3 experiments each lasting 14 days.

In experiment 1 : Twelve pigeons were divided into 2 groups of 6 birds each. One group received 0.5 ml of the virus suspension, and the other was left as control.

In experiment 2 : Thirteen birds were divided into three groups. Four birds were inoculated with 0.25 ml of virus suspension orally, 5 pigeons received the same dose intramuscularly, and 4 birds were left as uninoculated controls.

In experiment 3 : Thirteen birds were divided into two groups. Nine pigeons were inoculated intravenously (0.25 ml per pigeon) with the virus suspension. Four pigeons were left as uninoculated controls.

In the three infected groups, pigeons were wing-banded and each group was placed in a wire cage. The control groups were kept in separate rooms. All of the birds were provided with feed and water *ad lib*.

The areas of infection were not investigated as in BEARD and EASTER's experiments using a type of Newcastle disease virus (NDV). Pigeons were observed daily for symptoms, and those which died during the experiment or were killed at the end were necropsied. Samples of visceral organs including liver, spleen, kidneys, brain, spinal cord, muscles and bursae were fixed in 10 % formalin, processed, sectioned and stained with haematoxylin and eosin (H & E).

## Results

The pathological responses of pigeons to inoculation with PPMV-1 are summarized in table I.

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Experiment number *	Route and dose of inoculation	Pigeon responses		
		Lesion	Infected **	Control
1	Oral	Mortality	4/6 (67 %)	0/6
	0.5 ml	Lesion	6/6 (100 %)	0/6
2	Oral	Mortality	1/4 (25 %)	0/4
	0.25 ml	Lesion	4/4 (100 %)	0/4
	Intramuscular	Mortality	4/5 (80 %)	0/4
	0.25 ml	Lesion	5/5 (100 %)	0/4
3	Intravenous	Mortality	6/9 (67 %)	0/6
	0.25 ml	Lesion	9/9 (100 %)	0/6

TABLE I Responses of pigeons to infection with the PPMV-I.

\* Experiments 1, 2 and 3 were terminated 14 days post inoculation. \*\* Number affected per total examined.

#### Clinical signs

Whatever the route of infection, the symptoms observed in pigeons were similar. The predominant signs included depression, coma and death. Shaking and noding and/or abnormal carriage of the head, muscular temors and paralysis of swings and legs frequently observed. Occasionally, eyelids were swollen, stick together and had serous discharge. Un-inoculated pigeons did not show such signs.

### Mortality

In each of the inoculated groups, mortality started 5 or 6 days *p.i.* and 1-2 pigeons died thereafter every day throughout the observation period. As seen in table I, doubling the oral route resulted in a significant death increase. Both intravenous and intramuscular routes caused high percentages of mortality. No mortality occurred in the inoculated birds (table I).

## Gross lesions

No significant difference was detected between the inoculated groups and the predominant lesions included congestion and swelling of visceral organs particularly liver, kidneys, spleen and pancreas. The blood vessels at the surface of the brain were conspicuous and subdural haemorrhage was seen in several cases. The pancreas had discoloration and sometimes tiny focal greyish areas were seen. Muscles were dark and dehydrated. Emaciation was noticeable in a few of the infected pigeons, especially those that died later or were killed at the end of experiments. These lesions were not detected in uninoculated control groups.

# Histopathology

The histopathological changes were confined mainly to the brain, kidneys, liver, pancreas, lungs and intestine by decreasing order ; the reaction of the nervous tissues to the virus was seen in the cerebrum, cerebellum, pons,

medulla, meninges and spinal cord. It was a non-purulent meningo-encephalo-myelitis, which varied from mild to very severe and involved both the white and the grey matter. Blood vessels were congested and faecal haemorrhage was evident (photo 1). Perivascular cuffing with mononuclear cells and diffuse and/or focal glial cell proliferation were prominent (photo 2). Vasculitis with proliferation of intima and partial or complete obliteration of the lumen were detected. Changes were associated with oedema, neuronophagia and acute ischaemic necrosis (photo 3). Demyelination was evident and vacuolations were particularly prominent in the white matter of the cerebellum. Fibrillary gliosis or areas of cell proliferation extending from the granular layer to the meninges were encountered in the cerebellum. Meningeal serofibrinous exudate which contained a few mononuclear cells was evident. Meningeal wall thickness reached a 5-6 cell layer in some areas.



Photo 1: Extravasation of red blood cells through blood vessel wall and haemorrhage (x 400).



Photo 2 : Brain focal and diffuse gliosis and severe neuronal degeneration (x 400).



Photo 3 : Brain perivascular cuffing and vaculation (x 40).

The changes in the spinal cord were similar to those of the brain and included diffuse and focal fibrillary gliosis, neuronal degeneration, neuronophagia and demyelination.

#### The liver

Necrotic hepatitis and fatty changes were the main features observed. The sinusoids were severely dilated. Hepatocytes were necrotic and cell cords atrophic. Fatty changes « in the form » of fine vacuolation was encountered throughout the liver parenchyma. Infiltration by lymphoid cells and/or heterophils were confined to portal regions.

The main changes in the kidneys were mild to moderate interstitial nephritis with infiltration by lymphoid cells and/or heterophils between tubules that had undergone degenerative changes (photo 4). They could be accompanied by haemorrhages and congestion of blood vessels. In a few cases, exudative or proliferative glomerulitis was evident.



Photo 4 : Kidney. Interstitial nephritis with lymphoid cell infiltration and tubular degeneration (x 400).

Mild to moderate pancreatitis was evident mainly in the glandular acinar tissue. Foci of vaculoation and/or oedema and reticular cell proliferation had variable sizes. Proliferation of lymphoid follicles, degeneration and atrophy of acinar cells and, to a lesser extent, islet cell degeneration were also noted.

The intestinal tract developed catarrhal enteritis with tiny focal haemorrhages as well as proventriculitis with increased mucus cells, oedema and serous infiltration with mononuclear cells.

The changes in the respiratory tract included bronchiolitis with serofibrinous exudate, increased mucus cells and sometimes epithelial hyperplasia. Alveolar vessels were distended with red blood cells and proliferation of a few lymphoid foci. Submucous oedema, mononuclear proliferation and congestion were seen in the trachea. Fibrinous pericarditis and mild serous myocarditis were rarely encountered.

## Discussion

Nervous manifestations seen with PPMV-1 can be confouned with those caused by herpes virus infection, salmonellosis, and severe parasitic infestation. Isolation and identification of the agent makes the final diagnosis. However, where isolation facilities are lacking or where there is a delay, a history of the disease, a complete picture of pathological alterations together with histology might aid in determining the differential diagnosis.

In the central nervous system, PPMV-1 in pigeons (9) and avian influenza virus (1) in chickens, produce mild to severe non-suppurating meningo-encephalo-myelitis characterized by gliosis, vasculitis and meningeal thickening.

Newcastle disease is seldom severe in pigeons in this country where the velvogenic strain is endemic. Avian influenza as a natural disease has not been reported (7) and even highly pathogenic strains for turkeys produce little effect in pigeons. In Newcastle disease in chickens vascular damage are prominent in the central nervous system.

Vasculitis with hyperplasia of the endothelial lining of blood vessels is an important finding in pigeons. Thus, it can be speculated that PPMV-1 also attacks the endothelial lining of blood vessels. Hyperplastic blood vessels between central folia and extending into the granular layer has also been reported for Newcastle disease in chickens (8).

The hyperplasia of cells of the parabronchial wall resulting in space obliteration and lung consolidation, or the fibroblastic consolidation of the septa seen in Newcastle disease in chickens (6) were not observed. PPMV-1 produced degenerative pancreatic changes in pigeons, but such changes were not reported in chickens with Newcastle disease virus. Only lymphocytic infiltrations

were reported in the pancreas. However, in the closelyrelated avian influenza virus, mild to moderate or severe necrotizing pancreatitis with intranuclear inclusions in the islet cells was produced by a highly pathogenic strain in chickens (1). Necrotizing myositis were encountered in infections due to PPMV-1 NDV and avian Influenza virus.

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Oral, intramuscular or intravenous experimentally induced infection of pigeons by paramyxovirosis serotype I (PPMV-1) resulted in nervous signs, and diarrhoea. Necropsic examination showed mild to moderate congestion of viscera and catarrhal enteritis. Histo-pathological changes were most frequent in the central nervous system, followed by kidneys, liver, pancreas, intestines and lungs, together with mild to severe non-purulent meningoen-cephalitis. Key words: Pigeon - Paramyxovirosis - Experimental infection -The Sudan.

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# Caprine arthritis-encephalitis antibodies in indigenous sheep in Saudi Arabia

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ALLUWAIMI (A.M.), ABU EUZEIN (E.M.E.), HASSANEIN (M.M.). Anticorps contre l'arthrite-encéphalite caprine chez des moutons d'Arabie Saoudite. Revue Élev. Méd. vét. Pays trop., 1990, 43 (4) : 444-445 Une étude sérologique a été menée chez des moutons d'Arabie Saoudite afin de rechercher des anticorps contre le virus de l'arthrite-encéphalite caprine ; 0,8 p. 100 seulement des sérums examinés étaient positifs. Des considérations sur l'épizootie de la maladie dans la région sont discutées. Mots clés : Mouton - Arthrite - Encéphalite - Sérum - Arabie Saoudite.

#### Introduction

Caprine arthritis-encephalitis (CAE), a disease usually affecting young goats, involves the central nervous system and connective tissues, especially those associated with the synovial lined cavities (4). Degenerative arthritis becomes chronic and is mostly seen in adults. Sick goats show acute articular swelling and pain. The main pathological features are synovial cell hyperplasia and infiltration by leucocytes. There is also proliferative synovitis of joints, tendon sheath and bursae, characterized by villous hypertrophy. Later stages lead to fibrosis, necrosis and mineralization of synovial membranes and periarticular collagenous structures leading to anchylosis (1, 4).

The causative virus of CAE belongs to the retroviridae family, the lentivirinae subfamily. This subfamily includes virus responsible for several slowly developing, often fatal, diseases in man and animals (7). The disease has been reported from various parts of the world (2, 6, 9).

As the Kingdom of Saudi Arabia imports live animals from various parts of the world, it is expected that some foreign diseases of domestic animals could be introduced into the country.

The present study was initiated by the observations that symptoms of swollen joints and anchylosis are often seen in sheep and goats at the Al-Ahsa oasis. Since repeated trials to isolate the CAE virus, or any other microorganism from such animals were unsuccessful, we carried out some experiments to look for serum antibodies against the CAE virus, as a preliminary guide-line for future studies of this disease.

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