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CLINICAL PAPERS

Paramyxovirus disease in racing pigeons
Clinical aspects and immunization. A report from the Netherlands

J. T. Lumeij¹ and J. W. E. Stam²

SUMMARY Since 1981 a highly contagious viral disease causing high morbidity and low mortality in racing pigeons has spread over Europe. The virus belongs to the avian paramyxovirus sero group I. Clinical signs include watery droppings, polydypsia and neurologic signs in a high proportion of infected animals. Definitive diagnosis can be made by virus isolation in cell cultures or chicken embryos, and virus identification by haemagglutination and haemagglutination inhibition (HI) tests. The HI test, using sera from suspected animals, is a useful clinical tool to confirm the diagnosis. The most important differential diagnosis is salmonellosis. Good immunity against this disease can be acquired by subcutaneous vaccination with an inactivated oil adjuvant poultry NDV-vaccine. For the benefit of pigeon racing a plea is made for compulsory vaccination in countries in which the disease is endemic.

INTRODUCTION
The avian paramyxoviruses have been divided into six serologically distinguishable groups, with the adoption of the nomenclature PMV-1 to PMV-6 to distinguish serotype (1). Although antigenic variation does exist, Newcastle disease virus (NDV) isolates are usually considered to be a serologically homologous group because of the close relationship seen in haemagglutination inhibition (HI) tests. NDV has been classified as PMV-1, and is the only representative of this serogroup (1). Until recently clinical infections with NDV in pigeons (Columbia livia) occurred sporadically, and only secondary to NDV-infections in poultry (6, 10, 13, 15, 16, 19).

In 1981 several outbreaks of Newcastle disease (ND) in pigeons characterized by low morbidity and low mortality were observed in the province of Emilia in the north of Italy. In poultry stocks no clinical signs were seen, leading to the supposed existence of a NDV strain highly specific for pigeons. The disease was characterized by neurologic signs. A pigeon specific neurotropic strain of NDV, which was not pathogenic for chickens, was isolated by Biancifiori and Fioroni from these cases (3, 20).

In 1983 Richter and Kösters described an infection in pigeons by a viscerotropic and neurotropic paramyxovirus which did not cause clinical symptoms in hens on the same premises in Oberbayern, Germany (14). In February 1983 a pigeon specific viscerotropic and neurotropic strain of NDV was isolated from an outbreak of ND in pigeons in Oost Vlaanderen, Belgium (17). In January 1983 a disease resembling ND was seen in racing pigeons in the Netherlands. Virus isolation and identification was carried out by the Poultry Health Department². The causative virus was classified as belonging to the serogroup PMV-1 on the basis of HI-tests.

Although clinical outbreaks were restricted to pigeons, experimentally induced infections in one-day-old chickens caused morbidity and mortality, depending on the inoculation method.

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In England paramyxovirus disease in racing pigeons (PMD-P) was observed for the first time in July 1983. In the latter country ND-outbreaks in poultry have been associated with PMD-P. Feedstuffs contaminated by droppings from feral pigeons have been incriminated as the cause of these outbreaks (2).

In Japan outbreaks of a disease in racing pigeons have been observed with signs very similar to PMD. As yet no data are available in relation to virus isolation and identification (5).

This paper reviews aspects of PMD-P considered to be of interest to the practicing veterinarian dealing with racing pigeons. The information presented is based on the experiences of the Small Animal Clinic, State University Utrecht, gained during the 1983 PMD-P outbreak in the Netherlands, supplemented with data from the literature.

TRANSMISSION

The following is based partly on the immense experience with ND in poultry (7). The disease may spread to healthy birds by direct contact or indirectly by inhalation of dust from litter, food stuffs, drinking vessels, nest bowls and utensils contaminated with secretions and excretions of infected pigeons. Spread is mainly associated with traffic in live birds, contact between pigeons from different owners in pigeon baskets, and the movement of people between clean and infected premises.

The spread of PMD-P in the Netherlands in February 1983 has been attributed to intensive contact between racing pigeons during a national pigeon exhibition (J. v. d. Sluys, personal communication). Numerous cases of PMD-P were seen in July and August 1983, when young unvaccinated pigeons came in contact with each other in transport vehicles before racing.

The disease can also spread as a result of inadequate disinfection of infected premises and transport vehicles. NDV of chickens, for example, has been recovered from hen houses, egg shells, feathers and other materials up to 8 weeks after depopulation following an outbreak of the disease. In chickens NDV is excreted up to 4 weeks after natural infection. This is probably also true for PMD-P. The virus can be transmitted by air over short distances. Feral pigeons (Columbia livia) can act as a reservoir for the disease (2).

CLINICAL SIGNS

Clinical signs can occur between one and three weeks after natural infection. The average incubation period is 10-12 days (17, 18, 19). Usually the first clinical sign in a flock of pigeons is the excretion of watery droppings in 20 to 70 percent of the unprotected animals. This symptom is accompanied by polydypsia. In a substantial portion of the pigeons these initial signs are followed by neurologic signs, including ataxia, tremor of the head and neck, torticollis varying from slight head tilt to carriage of the head upside down (Fig. 1), falling to one side, paresis or paralysis of one or more extremities. A typical sign of atactic pigeons is their inability to pick up a single grain of corn from the floor.

Some pigeons only have watery droppings and polydypsia without showing neurologic signs. The reverse is also possible. In adult pigeons mortality is negligible. Although partial or complete recovery is possible in adult pigeons with neurologic signs, a high percentage of affected pigeons are unsuitable for racing.

In contrast to adult pigeons, affected squabs show a high mortality. A striking difference with ND in chickens is the occurrence of respiratory symptoms in the latter.

1 Racing Pigeon Clinic, Straatweg 5, 3621 BG Breukelen, the Netherlands.
DIAGNOSIS

No specific macroscopic lesions visible at post-mortem examination have been described.

Histologically a non-purulent encephalitis has been described (17).

The virus can be isolated from tissues of affected animals in cell-culture or chicken embryos and identified by haemagglutination (HA) and HI-tests. The HI test, using paired sera from suspected animals, is a useful clinical tool to confirm the diagnosis PMD-P.

DIFFERENTIAL DIAGNOSIS

PMD-P must be differentiated from a number of other diseases which can cause similar signs. The most important disease to be considered is salmonellosis, caused by *S. typhimurium var. copenhagen.*

In contrast to PMD-P, salmonellosis in adult pigeons does not as a rule manifest the characteristics of an acute infection. Usually in salmonellosis only a small number of birds show clinical signs. Infection may spread within a flock for a long period of time without producing distinct signs.

Different organ systems can be affected in salmonellosis. Common forms recognized in pigeons are the enteric form, the arthritic form and the neurologic form. Different pigeons in a flock may show different forms of the disease.

In the enteric form abnormal droppings are greenish and mucoid and not as watery as in PMD-P. Emaciation is commonly seen as an accompanying sign in salmonellosis, but not in PMD-P.

In the neurologic form the signs are indistinguishable from PMD-P. The percentage of affected pigeons however, is much less in salmonellosis. A symptom very commonly seen in PMD-P is that a number of pigeons from a flock are unable to pick up corn from the floor because of coordination disturbance. This sign is seen sporadically in salmonellosis and only in individual birds. Lameness due to salmonellosis is caused by arthritic changes which can be clinically detected as joint swelling. The elbow is most often affected. Swelling of this joint can be considered almost pathognomonic for salmonellosis and has never been observed in PMD-P.

Knowledge of the vaccination status of the animal with regard to both salmonellosis and PMD-P can help in diagnosis.

A suspected case of salmonellosis can be confirmed by faecal culture or serologic examination for agglutinating antibodies against formalin inactivated strains of *S. typhimurium var. copenhagen.* Pigeons which have been vaccinated can however show false positive titres. Squabs often die in the shell during the last days of incubation or shortly after hatching if maternally infected. Squabs that become infected with salmonella after hatching show retarded growth and lethargy. They may show malabsorption of the crop content and diarrhoea. Extreme emaciation is common. In advanced stages neurologic signs are frequent.

To complicate matters, we have observed a number of cases in which PMD-P was superimposed on salmonellosis. Other conditions to be considered in the differential diagnosis are parasitic infection and intoxication.

Trichomoniasis is a well known cause of mortality in squabs. Nematodes and coccidia can cause diarrhoea in adult pigeons. Diarrhoea and mortality may be caused by *Spironucleus columba* infection in squabs and young pigeons (21). A wide variety of environmental pollutants and even therapeutics can cause diarrhoea or neurologic signs. Mortality of squabs can also occur in pigeon lofts where the temperature becomes too high.

A syndrome which occurs in adult pigeons feeding their young should also be considered; this occurs when the squabs are about 8 days old and is characterized by profuse diarrhoea and polydypsia in the adult pigeons. Several breeding pairs can be affected at the same time. As yet no cause has been established.

Pigeon Herpesvirus (PHV) infections have been described as the cause of neurologic signs in pigeons (11) and should be considered in the differential diagnosis.

Although PHV is common in Belgium,
France and Germany (9), PHV from racing pigeons in the Netherlands has not yet been isolated, nor have antibodies against this disease been demonstrated.

VACCINATION

When in February 1983 PMD-P started to spread over the country, the Dutch Homing Pigeon Fanciers Association (Nederlandse Postduivenhouders Organisatie) made vaccination of its birds against the disease compulsory. In March 1983 approximately 3 million racing pigeons were vaccinated with one of the three permitted inactivated oil-adjuvant vaccines for NDV (0.20-0.25 ml subcutaneously in the neck).1,2,3

This results in a decreased incidence of PMD-P, followed by a reappearance of cases after 3 months (only in unvaccinated pigeons, however). Even if there was intensive contact between diseased and vaccinated pigeons, the latter remained free of clinical symptoms. Clinical cases were confirmed by virus isolation from tissues from pigeons sacrificed, or by HI-tests in serum, at the Poultry Healthy Department. We observed no field cases of PMD-P within 6 months of primovaccination with one of the vaccines mentioned.

Our clinical impression is substantiated by the vaccination trials performed by Viaene et al (18), who concluded that subcutaneous injection with an inactivated NCD oil adjuvant vaccine protected pigeons against intranasal and conjunctival infection with PMD-P for at least six months. This means that a single subcutaneous injection is sufficient to guarantee protection during the racing season.

At the Poultry Health Department it has been found that immunity after a second vaccination lasts even longer than 6 months. This is probably due to a booster effect.

Untoward side effects are sometimes observed after vaccination with inactivated oil-adjuvant ND vaccine administered subcutaneously. The most serious complication is mortality. Birds collapse within a few hours of vaccination and die within a few hours (22). The incidence of this complication has been reported to be 1 in 1,000 (4); in our experience acute mortality is 1 in 3,000.

It has been stated that the administration of betamethasone (0.25 ml in the breast muscle) may be an effective treatment in this situation (22). When corticosteroids are administered immediately after vaccination, however, it is to be expected that the immune response will be insufficient. Another side effect is nodule formation at the site of injection. The nodules develop in 1 to 2 weeks. In some pigeon lofts the incidence may be as great as 20%, while in the majority of lofts this complication is not seen. Nodule formation is often accompanied by depression. Nodule formation may be caused by non-sterile or faulty injection techniques.

The vaccine should be administered subcutaneously (Fig. 2). When the vaccine is administered intramuscularly into the breast muscle, necrosis at the injection site will invariably result. A foreign body reaction or an allergic reaction to substances present in the vaccine, however, can not be excluded as a cause of nodule formation.

It has been stated that nodule formation is more common following second vaccination than first vaccination (8). Nodules usually disappear spontaneously. In selected cases persistent nodules may be surgically removed.

1 Delvax NCD Emulsion, Lab, Dr. de Zeeuw, Gist-Brocades, De Bilt, the Netherlands.
2 Newcavac Nobilis, Gist-Brocades Animal Health, De Bilt, the Netherlands.
3 Poulvac ND, Duphar, Amsterdam, the Netherlands.
When pigeons are vaccinated during moulting, this process may be retarded and abnormalities may develop in the large flight feathers. We have seen this phenomenon only in older pigeons vaccinated for the second time. In a number of cases the birds suffered from concurrent infection with Salmonella typhimurium or endoparasites and showed general malaise.

For this reason faecal samples should be routinely examined for nematode ova and coccidia, and when symptoms suggestive of salmonellosis are present the birds should be examined for this disease. Existing infectious diseases should be treated before vaccination is attempted.

If practicable older pigeons should be vaccinated after the moulting process is complete i.e. usually after 15 December. Young pigeons should ideally be vaccinated at 10 weeks of age but certainly not younger than 6 weeks. Pigeons should be vaccinated 4 weeks before contact with pigeons from other lofts during racing or exhibitions.

Breeding pairs should be vaccinated 4 weeks before coupling. Experimental findings have demonstrated that a single vaccination with a lentogenic NDV-strain (La Sota) by intranasal drop combined with conjunctival sac instillation (drop-method) did not prevent the occurrence of clinical symptoms and neurotropic localization of PMV (17, 18). Only after repeated vaccination 16 days after primovaccination was protection against PMV obtained with the drop-method; immunity lasting longer than two months has not yet been demonstrated.

The experimental findings imply that the drop-method should be performed 4 times to guarantee protection during the racing season.

Because in the Netherlands only veterinary surgeons are allowed to vaccinate pigeons against PMD, application of the ‘drop-method’ would be disproportionately expensive. Although the use of the ‘drop-method’ using La Sota vaccine has been advocated (4), at the present time there is no basis to justify further routine use of this method to achieve reliable protection against PMD-P. Only in exceptional cases will the ‘drop-method’ be applicable.

To prevent infection of PMV-free pigeon lofts, unprotected birds should not be vaccinated at a central point (club meeting etc.); the veterinary surgeon should visit each pigeon loft individually and take appropriate measures to prevent iatrogenic transmission of the disease.

LEGAL CONSIDERATIONS

The Dutch Homing Pigeon Fanciers Association was the first organization that made vaccination of its birds against PMD-P compulsory (March 1983). For obscure reasons this policy has not been continued in 1984. At the present time (July 1984) vaccination against PMD-P is done on a voluntary basis in the Netherlands.

Only pigeon fanciers who wish to participate in racing from England to the Netherlands have to prove that their pigeons have been 'fully vaccinated' (see below). Vaccination of pigeons against PMD-P in the Netherlands may be performed only by veterinary surgeons (Wet op de Uitoefening van de Diergeneeskunde, Art. 6, lid 1). As long the Dutch poultry export is not threatened because of ND infection in poultry, the Dutch government will not make vaccination of pigeons against PMD-P compulsory. It is unlikely that outbreaks of ND in poultry will occur in the Netherlands because the entire poultry population is vaccinated against ND.

The German Homing Pigeon Fanciers Association (Verband Deutscher Brieftaubenliebhaber) has made vaccination against PMD-P obligatory for those members who want to participate in racing. The use of only one vaccine, based on inactivated virus¹ is permitted (8, 12). When foreign pigeons are imported into Germany they must be vaccinated with inactivated oil adjuvant ND-vaccine. In England the current position is that unvaccinated pigeons may not enter, leave or pass through an area in which movement is restricted because of PMD in pigeons or ND in poultry. Vaccinated pigeons however, may move in an area in which PMD-P has been confirmed.

Vaccination against PMD-P is compulsory

¹ Newcavac Nobilis, Gist-Brocades Animal Health, De Bilt, the Netherlands.
for continental pigeon fanciers who wish to race their pigeons from England to the Continent. Pigeons must be ‘fully vaccinated’ (i.e. twice, with an interval of at least 14 days) with an inactivated oil adjuvant ND vaccine administered subcutaneously. Because in England vaccination of poultry against ND is not practised as it is in the Netherlands, and because ND-outbreaks in poultry have occurred secondary to P-MD-P, legislation concerning P-MD-P is very strict in this country. In England and Germany attenuated live ND-vaccines in pigeons are not approved for vaccination against P-MD-P.

CONCLUSIONS-
PMD-P in racing pigeons is an important infectious disease with high morbidity and low mortality. Reliable protection against this disease during the racing season can be acquired by subcutaneous administration of an inactivated oil-adjuvant poultry NDV-vaccine. The best time to vaccinate adult pigeons is after moulting (from 15 December onwards) and before breeding. Young pigeons should be vaccinated 4 weeks before racing. Complications from vaccination can be minimized by using the described technique.

Revaccination of adult pigeons between 15 December and the end of January is necessary. Pigeon racing in countries in which P-MD-P is endemic would benefit from an obligatory vaccination with inactivated oil-adjuvant NDV-vaccine, using the vaccination scheme described. If the authorities do not make vaccination compulsory, it is clear that the National Homing Pigeon Fanciers Association concerned must take appropriate action.

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