

## CLINICO-PATHOLOGICAL OBSERVATIONS OF PIGEONS (*COLUMBA LIVIA*) SUFFERING FROM NEWCASTLE DISEASE

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### ABSTRACT

A survey was conducted to study clinical signs, gross and histopathological lesions in pigeons with naturally occurring Newcastle disease. For this purpose, 30 pigeon lofts were visited. Among these, 14 lofts showed clinical signs of Newcastle disease, including mainly greenish white mucoid diarrhoea and nervous signs with high morbidity and mortality. Postmortem examination of affected birds showed lesions mainly in brain, liver, kidneys and spleen. Amongst various organs, kidneys were more frequently involved. Histopathological changes were also observed in lungs, liver, kidneys, brain and spleen. The results showed that the Newcastle disease virus was widespread in pigeons locally and caused heavy mortality. No preventive measures or vaccination is being adopted by pigeon fanciers to control the disease.

**Key words:** Pigeons, Newcastle disease, clinical signs, lesions.

### INTRODUCTION

Newcastle disease (ND) is world wide in distribution and has a wide range of hosts like chickens, pigeons, turkeys, partridges, pheasants, doves, sparrows, gees, starlings and other free flying birds (Vindevogel *et al.*, 1972). It is caused by avian serotype-1 paramyxovirus in pigeons that is closely related to paramyxoviruses causing ND in poultry but not identical (Alexander *et al.*, 1985). The disease in pigeons is characterized mainly by sudden onset of listlessness, in-appetence, nervous manifestations and inability to fly. Morbidity and mortality averages 100 and 80%, respectively (Eisa and Omer, 1984). Several outbreaks have been reported in pigeon lofts throughout the world including Sudan (Eisa and Omer, 1984), Continental Europe and Great Britain (Alexander *et al.*, 1984), India (Mangat *et al.*, 1988; Singh *et al.*, 1989), Germany (Fischer, 1986), Turkey (Coven *et al.*, 1999) and Japan (Maeda *et al.*, 1987). A scarce information on clinical signs, gross and histopathological lesions in pigeon Newcastle disease in Pakistan is available. This manuscript describes these aspects in pigeons.

### MATERIALS AND METHODS

A survey of Newcastle disease (ND) was carried out in 30 pigeon lofts selected randomly, out of which 14 were clinically affected. None of the lofts was vaccinated against ND virus. Clinical signs of ND were recorded. Postmortem examination of pigeons died of ND was conducted and gross lesions were recorded.

Morbid samples from brain, liver, kidneys lungs and spleen were collected and fixed in 10% buffered formalin for histopathological examination. These tissues were embedded in paraffin, sectioned and stained with haematoxylin and eosin (Bancroft and Stevens, 1990).

Blood smears stained with Wright-Giemsa's stain (Benjamin, 1978) were studied for protozoon infestation to differentiate between ND signs. Intestinal contents of all ND suffering pigeons were also examined under microscope for protozoa and other parasitic eggs. The data thus collected were subjected to Chi-square analysis (Steel and Torrie, 1980).

### RESULTS

Thirty pigeon lofts were visited, out of which 14(46.7%) showed clinical signs of Newcastle disease. Surveys indicated that the onset of ND in pigeons was sudden. Affected pigeons showed in-appetence, listlessness and greenish white diarrhoea. Morbidity was between 30 and 90% and mortality was between 30-100%. The order of clinical signs is summarized in Table 1. Diarrhoea, the most common clinical sign observed, was present in 92.86% of the affected pigeons, followed by nervous signs (28.57%), shivering (21.43%) and paralysis of legs and wings (7.14%). Respiratory signs were not observed in any of the pigeon lofts in the present study.

Body condition of pigeons affected by ND was satisfactory in majority of the cases (71.43%), however, 28.57% of the affected pigeons were emaciated. Postmortem examination of two dead or clinically

affected pigeons per loft was conducted which showed gross lesions in various organs. Table 2 shows that kidneys involvement was more frequent than other organs. Kidneys were having necrotic spots. Atrophy of kidneys was seen only in one bird. Colour of liver was lighter in one pigeon, while in another pigeon small areas of necrosis were seen. Spleen in one pigeon was only slightly enlarged. Heart lesions included pinpoint haemorrhages and slight enlargement. A few pinpoint haemorrhages were present in proventriculus of one bird. Blood vessels were congested and slightly swollen. No gross lesions were detectable in other organs including trachea, lungs, oral cavity, esophagus, gizzard and intestines.

**Table 1: Frequency of clinical signs in Pigeons naturally affected with Newcastle disease (n = 28)**

Clinical signs	Number	Percentage
Diarrhoea	26	92.86 <sup>a</sup>
Nervous signs	8	28.57 <sup>b</sup>
Shivering	6	21.43 <sup>b</sup>
Paralysis of legs and wings	2	7.14 <sup>c</sup>

Values with different superscripts differ significantly ( $P < 0.01$ ).

Histopathologically, there was extensive mononuclear cell infiltration including mainly lymphocytes, followed by monocytes and a few macrophages in intertubular spaces in kidneys indicating characteristics of interstitial nephritis in pigeons died of ND. Necrotic and degenerated cells were also present in some of the tubules.

Mild perivascular cuffing mainly with lymphocytes was observed. Vacuolar degeneration was present in hepatocytes suggestive of mild degree of damage to liver tissue. Up to 30% of lymphoid nodules in spleen were enlarged but maintained normal structure and pattern. Enlargement was mainly due to proliferation of lymphocytes suggestive of reactive nodules. Varying

**Table 2: Postmortem lesions in pigeons (n=28) showing gross lesions of Newcastle disease**

Organs	Number	Percentage
Kidneys	04	14.29
Liver	02	7.14
Spleen	01	3.57
Heart	02	7.14
Proventriculus	01	3.57
Brain	02	7.14

degree of mononuclear cells infiltration, mild to extensive, was also present in meninges, characteristic of non-suppurative meningitis.

Small to large multiple haemorrhages were seen in lungs parenchyma. Mild degree of mononuclear cell infiltration was present. However, no histological changes were detected in trachea of pigeons died of ND.

Intestinal contents of pigeons were examined for protozoa and other parasites as differential diagnosis for diarrhoea. No protozoa were seen during wet smear examination of intestinal contents. Blood smears were also examined for protozoa and parasites. No protozoa or parasites were detected in blood smears.

## DISCUSSION

Newcastle disease is a serious problem in pigeons. Fairly high morbidity and mortality rate due to paramyxovirus-1 (PMV-1) infection was observed in pigeons in the present study. Mangat *et al.* (1988) reported 50 to 70% morbidity in an outbreak of PMV-1 in racing pigeons in India. They further reported mortality around 60% in field outbreaks.

In the present study, greenish white diarrhoea and nervous signs were more frequently observed. Barton *et al.* (1992) also observed diarrhoea and nervous signs associated with ND in racing pigeons. Khan (1968) reported paralysis of legs and wings in 80 and 60% experimentally infected pigeons, respectively. The occurrence of paralysis and nervous signs reported by Khan (1968) are fairly high than those observed in the present study. Fischer (1986) observed nervous signs in 86% cases, diarrhoea in 18% and paralysis of legs and wings in 9% of naturally affected pigeons. Many other workers have reported similar signs of ND in pigeons in experimental (El-Mubarak *et al.*, 1990; Mishra *et al.*, 2000) and field outbreaks (Eisa and Omer, 1984; Tangredi, 1985; Fischer, 1986; Mangat *et al.*, 1988).

No respiratory signs were observed in the present study. Khan (1968) did not observe any respiratory signs in field outbreaks or experimentally inoculated pigeons but laboured breathing and respiratory distress was reported in pigeons kept in a cage adjoining experimentally infected pigeons.

In the present study, 71.43% affected birds showed satisfactory body conditions, while 28.57% birds were emaciated. However, it appears that body condition relates to the duration of the disease. Alexander *et al.* (1984) reported that general loss of condition and anorexia occurred occasionally in ND infected pigeons.

According to Herdt and Devriese (2000), general condition of the pigeons remains well in ND.

Gross lesions were present only in few cases in the present study (Table 2). One lesion in the affected pigeons was involvement of brain which showed swelling and congestion of blood vessels. This is in congruence with Khan (1968), who reported haemorrhages in the brain of birds infected with ND.

In this study lesions were also recorded in liver, spleen, kidneys and proventriculus. In most cases no gross lesions were recorded. Eisa and Omer (1984) and Maeda *et al.* (1987) reported no gross lesions in natural outbreaks of ND in pigeons, while El-Mubarak *et al.* (1990) reported congestion of viscera and catarrhal enteritis in experimentally infected pigeons with paramyxovirus-1. Catarrhal enteritis was also seen in the present study. Mubarak (2000) observed gross lesions and congestion in visceral organs in experimentally infected pigeons with chicken origin ND virus. Solyom *et al.* (1984) reported hyperaemia and swelling of the kidneys in natural infection. Mangat *et al.* (1988) reported changes in thymus and bursa of Fabricius which were not observed in the present study.

Histopathological changes were present in spleen, lungs, kidneys, brain and liver of the pigeons affected with ND in the present study. Hyperplastic changes in spleen in up to 30% of nodules appeared reactive to pigeon paramyxovirus-1 in field infection. This is in line with Mubarak (2000), who observed hyperplasia of lymphocytes in spleen of pigeons challenged with chickens ND virus. Changes in kidneys seen in the present study are in line with those reported by Barton *et al.* (1992). The predominant histological lesions in kidneys in their study were interstitial nephritis and chronic tubular necrosis. Nephritis was also observed by Mangat *et al.* (1988) but they did not classify type of nephritis. Fischer (1986) also observed mononuclear interstitial infiltration in kidneys in nearly all infected birds.

Mild degenerative changes were observed in liver in the present study. Fischer (1986) also observed mononuclear interstitial infiltration in liver in nearly all infected birds. However, Mangat *et al.* (1988) and El-Mubarak *et al.* (1990) observed hepatitis.

Although lungs appeared normal grossly, histological changes were detected including haemorrhages and mononuclear cell infiltration. Mubarak (2000) also observed haemorrhages and leucocytic infiltration in lungs but the type of leucocytes was not mentioned. Fischer (1986) did not observe haemorrhages but mononuclear interstitial infiltration was often seen in lungs of pigeons in natural

outbreaks of paramyxovirus-1. No histological changes were detected in the trachea.

Changes in brain seen in the present study are in line with those reported by Barton *et al.* (1992). The predominant histological lesions in their study were focal non-suppurative encephalitis. Brain lesions seen in the present study differ from those reported by Mubarak (2000), who reported liquefactive necrosis that was suppurative in nature in pigeons experimentally infected with chicken ND virus. In the present study, non-suppurative inflammatory changes were recorded. El-Mubarak *et al.* (1990) also observed a non-purulent meningo-encephalitis in pigeons experimentally infected with pigeon paramyxovirus-1.

It remains to be confirmed whether chicken ND virus causes liquefactive necrosis in pigeons. Fischer (1986) also observed only non-purulent panencephalitis and encephalomyelitis in 100% of the field outbreaks of paramyxovirus-1 infection in pigeons. Neuron degeneration, neuronophagia, gliosis, endothelial cell proliferation and severe degeneration of purkinje cell reported by Mangat *et al.* (1988) in field outbreaks were not seen in the present study.

Paralysis of wings and legs in the present study and reported by many other workers stated above could be due to lesions of peripheral nerves. Fischer (1986) observed mainly degenerative lesions in peripheral nerves in 46% of the cases. These degenerative lesions accompanied inflammatory lesions of the nervous system. It is concluded that the ND virus was wide spread in pigeons locally and no preventive measures or vaccination is being adopted by pigeon owners to control the disease.

## REFERENCES

- Alexander, D. J., G. W. C. Wilson, J. A. Thain and S. A. Lister, 1984. Avian paramyxovirus type-1 infection of racing pigeons: 3. Epizootiological considerations. *Vet. Rec.*, 115: 213-216.
- Alexander, D. J., P. Russel and G. Parsons, 1985. Antigenic and biological characterization of avian paramyxovirus type-1 isolates from pigeons: An international collaborative study. *Avian Pathol.*, 14: 365-376.
- Bancroft J. D. and A. Stevens, 1990. *Theory and Practice of Histological Techniques*. Churchill Livingstone, London, UK.
- Barton, J. T., A. A. Bicford, G. T. Cooper, B. R. Carlton and C. J. Cardona, 1992. Avian paramyxovirus type-1 infection in racing pigeons in California. *Clinical signs, pathology and serology. Avian Dis.*, 36: 463-468.

- Benjamin, M. M., 1978. Outline of Veterinary Clinical Pathology. 2<sup>nd</sup> ed. Iowa State Univ. Press, Iowa, USA.
- Coven, F., D. J. Alexander, O. Z. Misirlioglu and I. Ozdemir, 1999. A study of paramyxovirus-1 infection in pigeons. *Bornova-Veteriner-Kontrol-ve-Arastirma-Enstitusu-Dergisi*, 24: 1-10.
- Eisa, M. and E. A. Omer, 1984. A natural outbreak of Newcastle disease in pigeons in Sudan. *Vet. Rec.*, 114: 297.
- El-Mubarak, A. K., E. E. E. A. Elzein, A. I. A. Elgasim, E. M. E. Abu Elzein and A. I. A. Elgasim, 1990. Note on the pathology of experimental infection of pigeons by the pigeon paramyxovirus type-1 (PPMV-1). *Revue-d'Elevage-et-de-Med. Vet.Des-Pays-Tropica*, 43: 441-444 (*Vet. Bull.*, 62: 1870, 1992).
- Fischer, E., 1986. Pathology of spontaneous paramyxovirus-1 infection in pigeons. Inaugural-Dissertation Fachbereich Veterinarmedizin Justus-Liebig-Universitat, Giessen, German Federal Republic, pp: 84 (*Poult. Abst.*, 13: 1917, 1987).
- Herdt, P.D. and L. Devriese, 2000. Pigeons. In: *Avian Medicine*, by Tully, T. N., M. P.C. Lawton, G. M. Dorrstein (Eds.) Reed Educational and Professional Publishing Ltd. Oxford, UK, pp: 330
- Khan, M. A., 1968. Epizootiology of Newcastle disease in wild birds. MSc. Thesis, Deptt. Microbiol., West Pakistan Agri. Univ., Lyallpur, Pakistan.
- Maeda, M., S. Koizumi and M. Yachi, 1987. Histopathological changes in Newcastle disease affecting racing pigeons in Japan. *Japanese J. Vet. Sci.*, 49: 217-223.
- Mangat, A. P. S., G. Singh and B. S. Gill, 1988. An outbreak of paramyxovirus encephalomyelitis in racing pigeons in India. *Vet. Rec.*, 123: 496.
- Mishra, S., J. M. Kataria, R. L. Sah, K. C. Verma and J. P. Mishra, 2000. Pathogenesis of Newcastle disease virus isolates in pigeon. *Indian J. Anim. Sci.*, 70: 1125-1126.
- Mubarak, A., 2000. Pathogenicity of Newcastle disease virus (chicken) in pigeons. MSc. Thesis, Deptt. Vet. Pathol., Univ. Agri., Faisalabad, Pakistan.
- Singh, G., A. P. S. Mangat and B. S. Gill, 1989. Isolation of Newcastle disease virus (avian paramyxovirus-1) from pigeons (*Columba livia*) in India. *Current Sci.*, 58: 154-155.
- Solyom, F., B. F. Nagy, M. Nemesi and E. Toth, 1984. Paramyxovirus infection in pigeons. *Allalteszegugyi es Takarmanvozas Kozlemenyet*, 20: 155-162 (*Poult. Abst.*, 12: 529, 1986).
- Steel, R.G.D. and J. H. Torrie, 1980. Principles and Procedures of Statistics. A Biometrical Approach. 2<sup>nd</sup> ed. McGraw Hill Book Co., New York, USA.
- Tangredi, B. P., 1985. Avian paramyxovirus type-1 infection in pigeons: clinical observations. *Avian Dis.*, 29: 1254-1255.
- Vindevogel, H., G. Meulemans, P. Halen and P. Schyns, 1972. Susceptibility of the adult carrier pigeons to Newcastle disease virus. *Ann. Res. Vet.*, 3: 519-532.