Vet.Med.J.,Giza. Vol.43,No.2.(1995):207-212.

PATHOGENICITY OF RECENT ISOLATES OF PIGEON PARAMYXOVIRUS IN PIGEONS IN EGYPT

By

EL-SISI, M.A.*; M. SHAKAL**; A. ALY*** and N.ABOU HASHEM*

* Department of Poultry Dis; Fac. of Vet. Med; Zagazig Univ.
** Department of Poultry, Fac. Vet. Med.; Cairo Univ, Giza,
** Department of Pathology, Fac. Vet. Med.; Zagazig Univ.

SUMMARY

Ninety pigeons of two months old were divided into six groups to study the pathogenicity of five PPMV-1 isolates with a control non-infected group. Each group was subdivided into three subgroups for intravenous, intranasal and contact infection. Clinical signs, morbidity, mortality and postmortem lesions are presented. Reisolation, seroconversion and histopathological changes are discussed.

INTRODUCTION

Mohamed et al., (1978, 1980) have recognised and described nervous manifestations in pigeons with highlosses and attributed the case to pigeon herps virus. In the past decade the disease problem had reached the panzootic form and was attributed to paramyxovirus-1. PMV-1 isolates from pigeons showed some antigenic variations from the classical NDV (Alexander et al., 1991).

In 1984 Eisa and Omar reported paramyxovirus infections in pigeons with sudden onset, rapid spread, and high mortality and morbidity. Pigeon fanciers in Egypt suffer from great losses due to outbreaks in pigeons diagnosed to be infected with the pigeon variant of paramyxovirus-1 (PPMV-1). A need to study to newly isolated PPMV-1thogenicity was raised. Pathological added to virological studies on the newly isolated PPMV-1 are performed in this work.

MATERIAL AND METHODS

Pigeons:

Ninty pigeons of 2 months old divided into 6 groups each of 15 were used for the experiments.

Fertile eggs:

Embryonated chicken eggs were inoculated per allantoic sac for pigeon paramyxovirus reisolation.

Viruses:

a- Pigeon paramyxovirus strain (715), this virus strain was used as an antigen for measuring the haemagglutination inhibiting antibody titer in experimentally infected contact pigeons.

b- Field isolates of pigon paramyxovirus identified by the authors were studied for their pathogenicity in pigeons.

Stains:

Haematoxylin and eosin were used for staining sections from parafin blocks of formaline fixed organ samples.

Experimental infection:

Ninety pigeons of 2 months old were divided into 6 groups of 15 pigeons each. Five groups were inoculated each with a recently isolated PPMV-1 from pigeons with nervous marifestation in Egypt. Each was subdivided into 3 subgroup of five pigeons, one subgroup for intravenous (1/V), one for intranasal and the last for contact infection. The infection dose for 1/v and intranasal was 10^8 EID_{50}/bird. The sixth group was kept as noninfected control. Clinical features, morbidity, mortality and postmortem lesions were followed up. Samples for virus reisolation and sera for measuring seroresponse were obtained. Brain, liver, kidneys and intestine were taken in 10% formaline saline for histopathological studies.

RESULTS

Clinical signs developed in all groups, showing diarrhea followed by nervous signs began on 3rd, 6th and 11th day postinfection for intravenous, intranasal and contact infected pigeons respectively.

Morbidity was 100% for all routes of infection. Meanwhile mortality rate was 100% in intravenous and intranasally infected pigeons and ranging between 60-80% in contact infection (Table 1).

Table (1): Results of experimental infection of different groups of pigeons with 5 different PPMV-1 isolates by intravenous, intranasal and by contact routes.

Isolate No.	Route of infection	Incubation period	Disease course	Morbidity %	Mortality %
I	I/V	3	13	100	100
	I/N	6	20	100	100
	contact	12	31	100	60
II	I/V	3	15	100	100
	I/N	6	19	100	100
	contact	15	31	100	60
III	I/V	4	8	100	100
	I/N	7	16	100	100
	contact	13	31	100	80
IV	I/V	4	12	100	100
	I/N	6	16	100	100
	contact	12	31	100	60
V	I/V	4	11	100	100
	I/N	6	20	100	100
	contact	13	31	100	60

I/V: Intravenous I/N: Intranasal

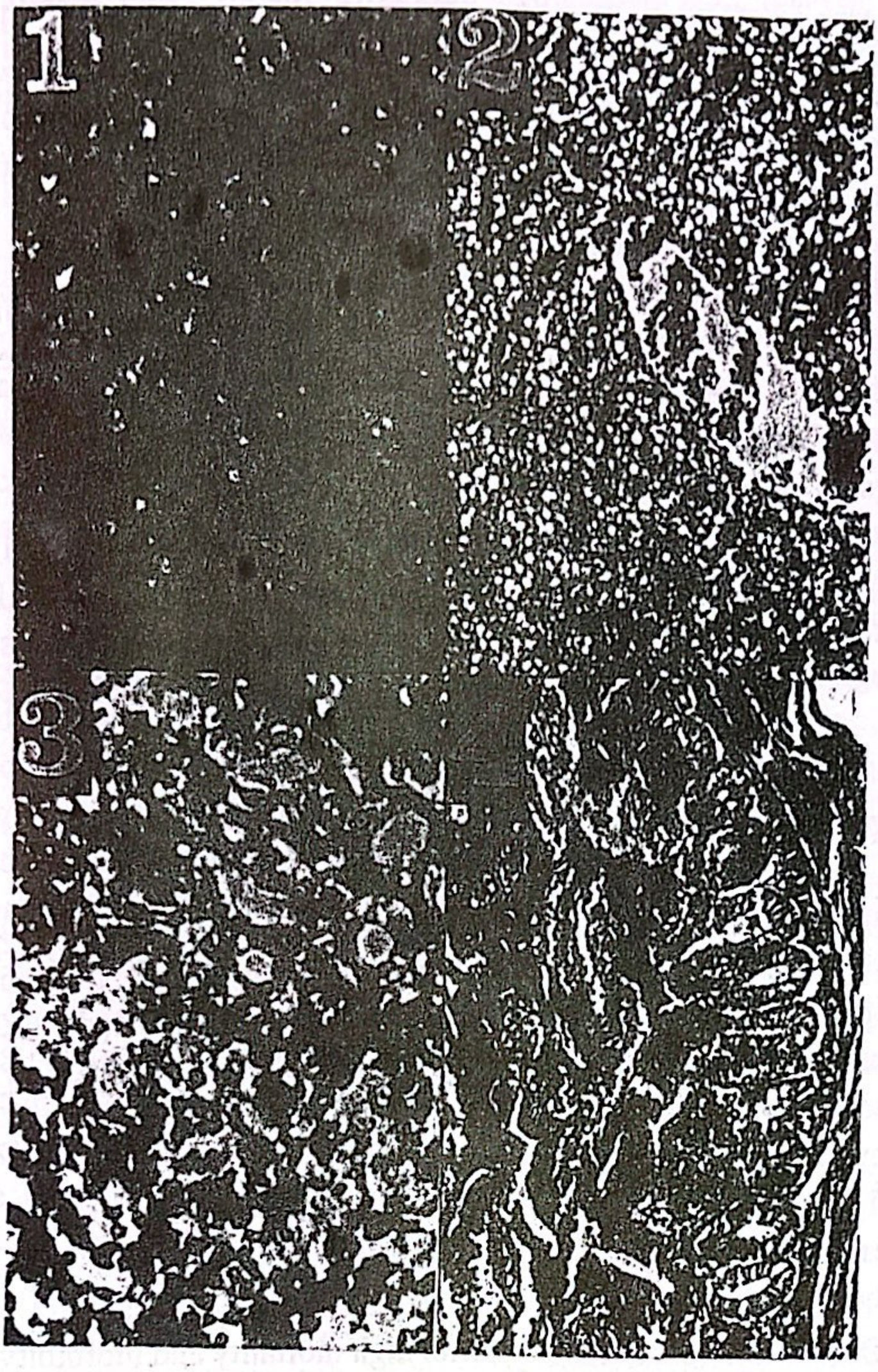

Fig (1): Brain of experimentally infected pigeon with PPMV-1 isolate showing degenerated neurons (A), satellitiosis (B) and neuronophagia.

Fig. (2): Liver of experimentally infected pigeon with PPMV-1 isolate showing fatty degeneration of the hepatic cells. H&E X 50.

Fig. (3): kidney of experimentally infected plgeon with PPMV-1 isolate showing focal replacement of renal medulla with lymphocytes (arrow) H&E X200.

Fig. (4): Intestine of experimentally infected pigeon PPMV-1 isolate showing desquamation of epithellal lining of intestingal villi and metaplasia of epithelial lining of intestinal glands to goblet cells (arrow). H&E X50.

Incubation periods after experimental infection were 3-4, 6-7, 12-15 days post intravenous, intranasal and contact infections respectively. While courses of disease were 8-13, 16-20 and 31 days, respectively.

Postmortem lesions were congestion and haemorrhages on brain and meninges, congestion with slight enlargement of visceral organs and enteritis seen in pigeons infected by any of the above mentioned routes. PPMV-1 virus was reisolated from all experimentally infected.

Heamagglutination inhibiting titers of sera collected from survivors 22 days post-contact ranged between 2^{7-8} against reference PPMV-1 Strain (715). No clinical signs, post-mortem lesion nor immune response against PPMV-1 were diagnosed in the noninfected control pigeons.

Histopathological changes:

Brain:

Satellitosis and neuronophagia were commonly seen in all examined brains in addition to focal areas of necrosis with perivascular oedema and lymphocytic cuffing around the submentingeal blood vessels (Fig. 1). Purkinje cells of cerebellum were degenerated and showed glial cell proliferations. Submeningeal blood vessels and cappillaries were congested.

Liver:

Degenerative changes were seen in hepatocytes and fatty changes characterized by numerous vacuoles of variable sizes pushing nuclei toward the periphery. Focal replacement of the hepatic tissues with lymphocytes particularly in the interstitial and subcapsular tissues (Fig. 2).

Kidneys:

Cloudy swelling and hydropic degeneration with desquamation of some epithelial cells into the luminae of renal tubules in both cortex and medulla. The intertubular blood vessels were congested and focal areas of coagulative necrosis were seen (Fig. 3). Focal replacement of some renal tubules in the medullary area with mononuclear cells including macrophages and lymphocytes. Epithelial lining of ureters showed hydropic degeneration and infiltration of the lamina propria with lymphocytes.

Intestine:

Metaplasia of epitelium lining of intestinal mucosa to goblet cells and infiltration of lamina propria with lymphocytes were seen in addition to congestion of blood vessels (Fig.4).

DISCUSSION

In Egypt, El-Dahaby and Sokkar (1967) have diagnosed natural ND in young pigeons. In contrast, Ahmed and Reda (1967) stated that pigeons are relatively resistant to natural infection but not to experimental one.

In 1984 Eisa and Omar reported that paramyxovius affections in pigeons were characterised by sudden onset and rapid spread with high mortality and morbidity.

Pigeon fanciers in Egypt are suffering from great losses due to paramyxovirus infections. Thereforhere was a need to study the pathogenicity of the newly isolated pigeon paramyxoviruses to locate their disease producing capacity in pigeons with spot lights on the histopathological changes due to the infection with this virus in pigeon.

Five recently isolated PPWV-1 induced classical signs in all groups of experimentally infected pigeons. Reisolation of the virus from pools of brains and visceral organs were successfull. Morbidity rate of 100% had been observed in intravenous, intranasal and contact nfection. Meanwhile mortality rates seen in intravenous and intranasal infection were 100%, contact infected birds had 60-80%. Mortaility, incubation periods, post experiemntal infection, and course of the disease induced were shortest in case of intravenous infection followed by the intranasal infection and longest in case of contact infection. Similar results were reported by Ballouh and El Zein (1986), whereas Pearson et al (1987) recorded no clinical signs in adult pigeons which had immunological response against the infection by contact.

In our experiments clinical signs began on the 3 rd day for intravenous, 6th day for intranasal and 16th day for contact infection. Delayed appearance of signs in contact infection may be attributed to the route of infection. Kascula (1951) stated that the inoculation site used has a direct bearing on symptoms observed.

The histopathological changes were mainly seen in brain followed by kidneys then liver. Those changes ranged between non-purulent encephalitis and degenerative changes in kidneys and liver with lymphocytic infitration. Fisher et al., (1986), Mangat et al., (1988), Sanford and Hampson (1989) and Barton et al. (1992) showed similar changes of paraffin sections in brain, kidneys and liver of experimentally infected pigeons.

As a conclusion from experiments, the recently isolated PPWV-1 showed high disease inducing capacity in pigeons, which requires secured vaccination programmes for pigeons in Egypt.

REFERENCES

Ahmed, A.A.S.; and Reda, I.M. (1967): Response of pigeons to eight strains of Newcastle disease virus. Avian Dis. 11,234-240.

Alexander, D.J.; Manvell, R.J.; Collins, M.S.; and Brockman, S.. (1991): Evaluation of relationships between avian paramyxoviruses isolated from the birds of family columbiae. Arch. Virol. 116, 167-276.

Ballouh, A.; and Abu-Elzein, E.M.E. (1986): Comparative pathogenicity of the "pigeon paramyxovirus 1" and five velogenic Newcastle disease viruses to chickens, chick embroys and pigeons in the Sudan. Bulletin of Animal Health and Production in Africa. 34 (4) 254-257.

Barton, J.T., Bickford, A.A.; Copper, G.L.; Charlton and Cardona, C.J. (1992): Avian paramyxovirus type infections in racing pigeons in Califomia-1 Clinical signs, pathology and serology. Avian Dis., 39; 463-668.

Eisa, M.; and Omar, E.A. (1984): A natural outbreak of Newcastle disease in pigeons in the Sudan. Vet. Rec. 114, 297.

El-Dahaby, H.; and Sokkar, I.M.H. (1967): Natural infection of Newcastle disease in Egyptian pigeon, J.Vet. Sc. UAR. 4 (11) 55-62.

Fischer, E.; Frese, K.; Meister, J.; and Kaleta, E.F. (1986); Histopathology of spontaneous PMV-1 infection of pigeons. Dt. Vet. Med. Gesellschaft, 141-149.

Kascula, V.R. (1951): An observation on the pathogenesis of artificial Newcastle dsease in pigeons, J.S. Afr. Vet. Med. Ass. 22, 195.

Mangat, A.P.S.; Singh, G., and Gill, B.S. (1988): An outbreak of paramyxovirus encephalomyelitis in racing pigeons in India. Vet. Rec., 123 (19) 496.

Mohamed, M.A.; Sokkar, S.M., and Tantawi, H.H. (1978): Contagious paralysis of pigeons. Avian Pathol. 7, 637-643.

Mohamed, M.A.; Sokkar, S.M.; Youssif, Y.I., Saad, F.; and Bastami, M. (1980): Contagious paralysis of pigeons. A preliminary investigation on the occurrence of the disease in Egypt. J. Egy. Vet. Med. Assoc., 40, (1) 45-51.

Pearson, J.E.; Senne, D.A.; Alexander, D.J.; Taylor, W.D.; Peterson, L.A.; and Russell , P.H. (1987): Characterization of Newcastle disease virus (Avian paramyxovirus-1) isolated from pigeons Avian Dis. 31 (1) 105-11.

Sanford S.E. and Hampson R.J. (1989): *Pigeon* paramyxovirus. Can Vet. J. 30 (6) 523.