

Original Research Article

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Newcastle Disease and their Pathology in Fowls Affected with Genotype XIII and Pigeons with Genotype II

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ABSTRACT

Chickens affected with Newcastle disease virus (NDV) genotype XIII and pigeons affected with genotype II were characterized for virulence and found to be velogenic and lentogenic respectively, based on protein translation of Fusion protein (F) gene and by Mean death time (MDT) and Intracerebral Pathogenicity Index (ICPI). Clinically, respiratory and/or enteric manifestations were exhibited by the chickens, whereas pigeons showed predominantly neurological signs. It was therefore pertinent to describe the salient pathological features or differences during spontaneous infections in the respective hosts. Disease was suspected in two flocks of backyard fowl (*Gallus gallus domesticus*) showing 100% morbidity and 86% mortality; and in six flocks of domesticated pigeons (*Columba livia domestica*) exhibiting 21.68% morbidity and 14.16% mortality in a study from August 2017 to July 2018. Gross lesions and microscopic lesions were typical for those described for ND, but viscerotropic lesions were prominent in fowls, while neurotropic lesions were more vivid in pigeons. Gross lesions were suggestive of vascular injury with haemorrhagic tracheitis, enteritis and sometimes petechiae observed in other organs. No prominent gross lesions were observed in the nervous tissue except in some birds showing congestion of meninges. Histologically, haemorrhages, mononuclear infiltration and lymphoid depletion were common. Lesions in nervous tissue were more pronounced in pigeons and represented focal gliosis, loss of Nissl substances and neuronal degeneration, satellitosis and neuronophagia. There was focal gliosis of the nerve tracts in the cerebellum indicating demyelination of nerve tracts. Consistent presence perivascular oedema, endothelial hypertrophy, necrosis and medial hyalinization were noteworthy. In some cases, vacuolation was observed in Purkinjee cells together with presence of intracytoplasmic inclusions. It is speculated that the relatively less pathogenic pigeon strain has sufficient time to generate an overt neurological lesion than the virulent fowl strain. The present findings highlight the salient differences in host pathology in chicken and pigeons and are believed to assist in diagnosis of the disease, particularly while attributing a particular pathotype to a prevalent strain.

Keywords

Fowl, Newcastle disease, Pathology, Pigeon

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Introduction

Newcastle disease (ND) is a contagious viral disease affecting many domestic and wild avian species and is considered to be one of the most important poultry diseases in the world. According to OIE (2012), Newcastle disease is a notifiable disease, and is characterized by high morbidity and mortality in poultry flocks that may reach 90-100%, depending on the variant strains. Apart from commercial and backyard poultry, a wide range of captive and free living birds are also susceptible, that can sometimes act as primary source of ND infection to chicken (Kouwenhoven, 1993; Alexander and Senne, 2008a). The disease has also been reported in pigeons that occurs due to the spread from diseased chicken flocks, and can occur vice versa from domesticated or feral pigeons to poultry (Alexander *et al.*, 1984).

By definition Newcastle disease virus (NDV) is a virulent virus (OIE, 2012) which is classified in the genus *Avulavirus*, all of which belonging to *Avian orthoavulavirus-1* (AOAV-1) group (Formerly APMV-1 or AA_vV-1) (ICTV, 2019). In pigeons, ND is highly contagious disease, caused by pigeon paramyxovirus serotype-1 (PPMV-1), a variant of APMV-1 causing ND in poultry.

The clinical disease forms vary, depending on various pathotypes designated as velogenic (viscerotropic or neurotropic), mesogenic and lentogenic in decreasing order of virulence. Depending on the pathotype and susceptibility of the host, the mortality varies from zero to 100% (Nanthakumar *et al.*, 2000). ND is endemic in India and different pathotypes of the virus have been isolated and characterized from several avian hosts. The disease has been documented in fowls (Sangha, 2017) and in pigeons (Mehmood, 2017) from Jammu; and also from chickens in Kashmir (Maqbool, 2016).

In a recent study, fowl and pigeon isolates from Jammu were characterized by deduction of protein motif of the Fusion (F) gene that governs virulence; also the pathogenicity was differentiated by illustrating the Mean death time (MDT) in eggs and Intracerebral Pathogenicity Index (ICPI) in day-old chicks wherein the fowl isolate was characterized as a velogenic strain, while the pigeon isolate was found to be a lentogenic strain (Chowdhary, 2018; Chowdhary *et al.*, 2020). Besides, it was also found by phylogenetic analysis that the fowl isolate clustered with genotype XIII, while the pigeon isolate grouped with genotype II. Since both isolates in the respective hosts were of different genotypes and pathotypes, it was intended to describe the differential and comparative pathology of the isolates in respective hosts during spontaneous infection.

Materials and Methods

Disease screening

Regular visits to localities around Jammu were made for outbreaks of suspected cases of Newcastle disease amongst rural backyard poultry, commercial broiler farms and pigeon coops. Materials also comprised of birds submitted to Division of Veterinary Pathology, for necropsy examination and avian disease diagnosis. The study was conducted from August 2017 to July 2018 for a period of approximately one year. Clinical signs and symptoms of birds suspected for Newcastle disease were observed and recorded during outbreaks.

Gross pathology

Detailed necropsy examination of birds were carried out and the lesions were recorded. Representative tissue samples were collected for visceral organs including lungs, trachea, heart, proventriculus, intestine, spleen, liver,

kidneys, cerebrum, cerebellum, and bursa of Fabricius.

Histopathology

Tissues were fixed in 10% neutral buffered formalin, processed for paraffin embedding, sectioned and stained with haematoxylin and eosin following standard procedure (Luna, 1968).

Results and Discussion

Prevalence of Newcastle disease in different birds

The disease was suspected in two flocks of backyard fowl (*Gallus gallus domesticus*) around Ranbir Singh Pura and Jammu; while amongst domesticated pigeons (*Columba livia domestica*) the disease was suspected in six flocks around Ranbir Singh Pura. The disease in fowls was particularly severe and presented with approximately 100% morbidity and 86% mortality. Amongst pigeons, the disease was relatively less severe with 21.68% morbidity and only 14.16% mortality.

Clinical signs of the disease in different birds

Fowls: Clinical signs and symptoms observed in the flocks were characterized by high mortality, with the younger birds earlier to succumb within 2-3 days of clinical manifestation. Affected birds showed lethargy and reduced activity, somnolence (Fig. 1A), inappetence, ruffled feathers, oedema of conjunctiva. Principal respiratory manifestation was dyspnoea and open mouthed breathing (Fig. 1B), swelling of the head, often with cyanotic discoloration. Birds excreted greenish or white watery diarrhoea. Some birds died suddenly with few or no signs.

Pigeons: Affected birds showed predominantly neurological symptoms. They included depression, mild to severe tremors, torticollis, in-coordination, unilateral or bilateral paresis, paralysis of wings, head pressing, circling and terminal coma (Fig. 1C-E). Birds with severe neurological signs often died due to inability to eat or drink. Sick birds were unthrifty and anorexic, often emaciated and dehydrated. Some birds were often found to void greenish-watery excreta (Fig. 1F). Affected birds if not dead, linger manifesting obvious signs for weeks; moderate recovery sometimes seen in few birds. Some birds may have been sub-clinically affected.

Necropsy examination and gross lesions

A total of 10 backyard poultry fowls (*Gallus gallus domesticus*) and 23 pigeons (*Columba livia domestica*) were assessed for detailed necropsy examination.

Fowl: Congestion and haemorrhagic lesions were one of the prominent visible lesions in most organs, particularly the respiratory and enteric system. There was widespread catarrhal or haemorrhagic enteritis. The crop was sometimes found to be distended, congested, and with a haemorrhagic mucosa. Haemorrhages were consistently present at the junction of the oesophagus and proventriculus and sometimes ulcerations in the intestinal mucosa and necrotic lesions on the serosa (Fig. 2A). Often haemorrhages were also noted in the ileo-caecal valve.

The lungs were almost always found congested, dark and oedematous. Severe congestion and haemorrhagic tracheitis was common (Fig. 2B). Cloudiness and fibrinous air sacculitis was seen in one case. Congestion and haemorrhages were also observed on the ovaries of adult birds (Fig. 2C), liver, spleen, epicardium (Fig. 2D). The kidneys and ureters were sometimes found to

be swollen with increased urate deposits. Meningeal congestion was marked (Fig. 2E) in two birds.

Pigeons: Necropsy examination often showed petechial haemorrhages on the mucosa of proventriculus with or without ulcerations (Fig. 2F). Congestion and haemorrhages were also noticeable on the crop. Petecheal haemorrhages were also seen on the duodenal mucosa of some birds (Fig. 2G) though haemorrhagic enteritis was not a consistent feature. In one case, a thickened and oedematous intestine was presented. Congestion and haemorrhages on the trachea (Fig. 2H) and lungs were seen in some birds. Occasionally, haemorrhages on pancreas, epicardium (Fig. 2I), congestion of meninges and brain (Fig. 2J), and liver were also recorded.

Histopathological lesions

Fowl: The observable lesions in the upper respiratory tract were found throughout the length of the trachea and bronchi of the lungs. Prominent lesions were severe congestion of the blood vessels, and thickening of the mucosa due to oedema and infiltration by inflammatory cells. The mucosal surface appeared to be undulant with variable thickening. Higher magnification showed deciliation and often sloughing of the mucosal epithelium. Lymphocytes and sometimes macrophages were found in large numbers invading the submucosa. The lungs appeared severely congested and consolidated. Oedema was evident in the parenchyma. These vascular lesions were distributed throughout the parabronchi and their alveolar areas. The bronchial areas reflected the lesions of the upper respiratory tract. There was considerable, but uneven thickening of the airway mucosa, sloughing and oedema. The mucosa also showed discontinuity in certain areas with presence of frank haemorrhages. A

variable degree of exudation was also present in the lumen of the airways and airspaces. Exudates comprised of cellular debris, fibrin, erythrocytes and mononuclear cells.

In the enteric system, congestion of the mucosa, oedema and infiltration were the predominant lesions. In the proventriculus, the mucosal vasculature were congested particularly in between the glands, and sloughing of the epithelium was sometimes seen (Fig. 3A). The glands were largely unaffected. Extreme congestion was sometimes seen in the mucosa, distending the villous structures. Ulcerations and haemorrhages were seen in some areas. The submucosa appeared thickened with cellular infiltration and proliferation of the smooth musculature. Lesions in the small intestines were often characterized by loss of mucosal epithelium, necrosis and sloughing. There was acute congestion of the villous structures resulting in their distension (Fig. 3B). Lesions in caecum were characterized by congestion, sloughing, haemorrhages and severe necrosis (Fig. 3C). A large number of mononuclear infiltrating cells were also seen in the submucosa.

The liver often appeared congested with focal or diffuse degenerative changes of the parenchyma. Vacuolation of the hepatocytes consistent with fatty changes were also sometimes seen (Fig. 3D). The kidneys showed degenerative changes of the distended tubular epithelium and presence of urates in their lumen. Oedema of the parenchyma was evident in some cases with distension of the intertubular spaces associated with mild degenerative changes of their epithelium.

In the heart, mild congestion and focal myocardial degeneration was sometimes noticed together with focal mononuclear infiltration in some areas. Microthrombi were also seen in some areas on the endocardium.

In the spleen, focal areas of necrosis and degeneration of lymphoid tissue, proliferation of macrophages and deposition of a homogenous material in the parenchyma, and also around the blood vessels (Fig. 3E) that appeared consistent with amyloid deposits. Depletion of lymphocytes and destruction of lymphoid follicles were also seen.

In the Bursa of Fabricius, a focal loss of lymphoid follicles was evident. The inter-follicular spaces were also increased with oedematous fluid. In some follicles, complete lymphocytic depletion and replacement with degenerate cellular debris and erythrocytes was seen. Lesions in the nervous system were very limited. In the cerebellum, focal gliosis could be seen in certain areas (Fig. 3F). There was also loss of Nissl substances in the Purkinjee cells. In the cerebrum, neuronal degeneration, satellitosis and neuronophagia could be seen (Fig. 3G). One consistent lesion was the hypertrophy of most vascular endothelium (reactive endothelium) (Fig. 3H), and the presence of perivascular oedema. Vascular changes also include medial degeneration and hyalinization, thrombosis in small vessels, and endothelial necrosis. Vacuolation and intracytoplasmic inclusions were apparent in some Purkinjee cells of the cerebellum (Fig. 3I).

Pigeons: Like in the fowls, lesions in pigeons were also very prominent in the respiratory tract. Moderate to severe congestion of the blood vessels and oedema was seen in throughout the length of the trachea. A large number of infiltrating mononuclear cells could be seen in the wall of the trachea. Hyperplastic changes were noticeable in the tracheal mucosal epithelium, marked by oedematous changes, proliferation of smooth muscle cells of the muscularis mucosa, and infiltration by mononuclear cells in the submucosa. The lungs appeared severely congested and consolidated (Fig. 4A).

Oedema was evident in the parenchyma. These vascular lesions were distributed throughout the parabronchi and their alveolar areas. Lymphoid proliferation of the bronchi associate lymphoid tissue was also notable, with profuse exudation in the airway lumen of the lungs. The bronchial wall also appeared oedematous with infiltration of mononuclear inflammatory cells.

In the enteric system, changes in the proventriculus included the congestion particularly in between the glands and severe oedema of the serosal layer (Fig. 4B). Severe congestion was seen in the mucosa, with occasional haemorrhages over the tips of the gland openings. Ulcerations and haemorrhages were seen in some other areas also. Lesions in the small intestines were often characterized by profuse mucus exudation, Goblet cell hyperplasia and serosal oedema (Fig. 4C). The liver appeared congested.

The kidneys showed profuse interstitial oedema, vacuolar degeneration of tubular epithelium, and presence of homogenous urate-like deposits in the tubular lumen (Fig. 4D). Changes in the spleen were not remarkable, and showed focal areas lymphoid follicular depletion, and erythrophagocytic macrophage aggregates in some areas.

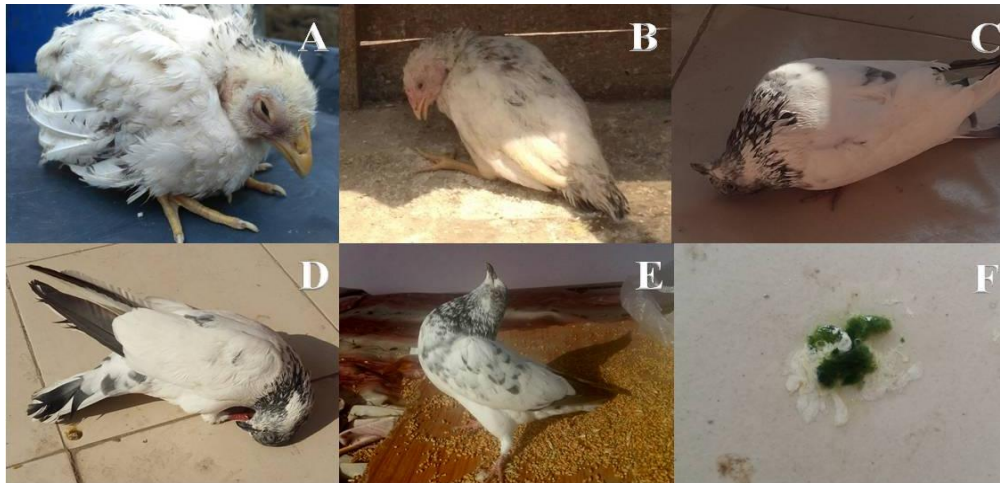
In the Bursa of Fabricius, a depletion of lymphoid follicles was very prominent. There was oedematous distension of inter-follicular spaces. Focal vacuolation and lymphocytic destruction in cortical areas and germinal centres were also visible (Fig. 4E).

Meningeal congestion and oedema was markedly noticeable. Focal gliosis could be seen in certain areas of the cerebrum (Fig. 4F). Satellitosis and neuronophagia particularly in the cerebrum was also noticed (Fig. 4G). Hypertrophy of the vascular

endothelium (reactive) and the presence of perivascular oedema was also noticeable (Fig. 4H). Similar endothelial necrosis and medial hyalinization were apparent. There was focal gliosis of the nerve tracts in the cerebellum

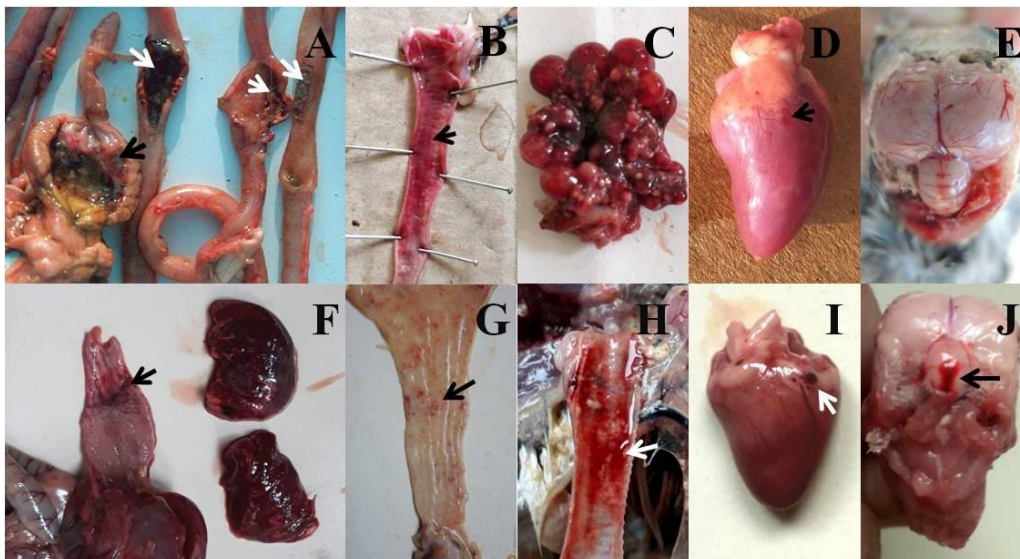
indicating demyelination of nerve tracts. Faint cytoplasmic vacuolation and intracytoplasmic inclusions were also seen in some Purkinjee cells of the cerebellum (Fig. 4I).

Fig.1 Clinical signs and gross lesions in birds suspected for Newcastle disease. *In fowls*



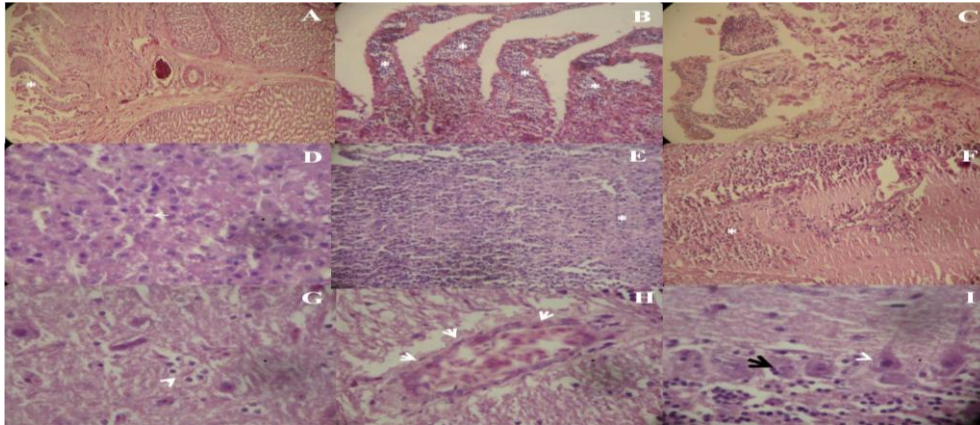
(A) Somnolence and lethargy (B) Laboured and open mouthed breathing; In pigeons: (C) Torticollis; (D) Head pressing; (E) Opisthotonus; (F) Greenish watery diarrhoea.

Fig.2 Gross lesions of affected birds suspected for Newcastle disease. *In fowls*



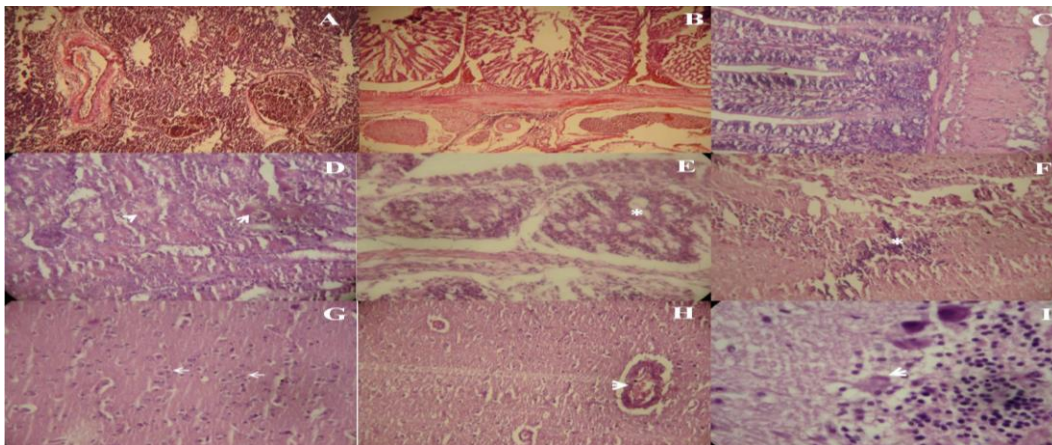
(A) Haemorrhages on proventriculus mucosa and intestinal ulcerations; (B) Haemorrhagic tracheitis; (C) Haemorrhagic oophoritis; (D) Petechiae on epicardium; (E) Meningeal congestion; In pigeons: (F) Haemorrhage at junction of oesophagus and proventriculus and severe congestion of lungs; (G) petechiae on duodenal mucosa; (H) Haemorrhagic tracheitis; (I) Epicardial petechiae; (J) Haemorrhage on meninges.

Fig.3 Histopathological enteric lesions of Newcastle disease in fowls



(A) Proventriculus showing congested vasculature and mucosal sloughing, H&E x 100; (B) Extreme congestion and distension of intestinal villi, H&E x 400; (C) Sloughing, haemorrhages and severe necrosis of caecal mucosa, H&E x 400; (D) Liver showing congestion and degeneration of hepatocytes, H&E x 100; (E) Necrosis and degeneration of lymphoid tissue, proliferation of macrophages and deposition of a homogenous amyloid-like material in the parenchyma, H&E x 400; (F) Focal gliosis in cerebellum, H&E x 100; (G) neuronal degeneration, satellitosis and neuronophagia in cerebrum, H&E x 400; (H) Hypertrophy of vascular endothelium, H&E x 400; (I) Cytoplasmic vacuolation of Purkinjee cells of cerebellum and presence of intracytoplasmic inclusions, H&E x 1000

Fig.4 Histopathological lesions of Newcastle disease in pigeons



(A) Severe congestion and consolidation of parabronchi of lungs, H&E x 100; (B) Proventriculus showing congested vasculature and serosal oedema, H&E x 100; (C) Goblet cell hyperplasia and mucous exudation in intestine, H&E x 400; (D) Distended kidney tubules with urate deposits, H&E x 100; (E) Focal vacuolation and lymphoid depletion in follicles of Bursa of Fabricius, H&E x 400; (F) Focal gliosis in cerebellum, H&E x 100; (G) Satellitosis and neuronophagia in the cerebrum, H&E x 400; (H) Hypertrophy of most vascular endothelium, and the presence of perivascular oedema in cerebrum, H&E x 100; (I) Intracytoplasmic inclusion in Purkinjee cells of cerebellum, H&E x 1000.

Sporadic outbreaks of Newcastle disease had always been suspected in local backyard poultry reared around the present study area, from cases that had been presented

infrequently for necropsy examination and disease diagnosis. The actual prevalence of the disease in fowls could not be ascertained due to the unorganized rearing in household

backyards. The disease epidemiology in pigeons was also limited because of the semi-domestic nature of rearing, where the actual flock size of the birds was not constant. There was intermingling of birds from neighbouring flocks, and even occasional ventures across the international Indo-Pakistan border in the immediate vicinity. Moreover, it is suspected the disease in many pigeons is subclinical and may have been overlooked. Reportedly, Newcastle disease in pigeons has a morbidity of 80% and mortality of 55% after experimental infection in pigeons (Biancifiori and Fioroni, 1983). The mortality rates as observed in the present study were far less, probably owing to the lowered virulence of the prevalent strain.

It is a well known fact that clinical signs of Newcastle disease are variable depending on the strain/ pathotype of the virus. Besides the host species, age of birds, immune status, stress factors, dose and route of virus exposure are important contributing factors (Alexander and Senne, 2008b). High mortality often upto 100% with virulent strains (VNDV) is reportedly typical in fowls, a fact validated by the present observation. Listlessness, increased respiration and weakness, ending with prostration and death have been corroborated in the literature with virulent virus strains in fowls (Alexander *et al.*, 1998; Balachandran *et al.*, 2014). As observed in the present study, other indicative signs like green diarrhoea and neurological signs including wing/ leg paralysis are reported to be indicative (Alexander and Senne, 2008b).

The disease observed in pigeons were also akin to descriptions in the literature. APMV-1 in pigeons is primarily associated with neurological signs similar to NDV in chickens (Heiden *et al.*, 2014). The clinical signs observed in pigeons were similar to descriptions of Marlier and Vindevogel

(2006) for the neurotropic form of NDV, such as tremor of the neck and wings, torticollis, paralysis and disturbed equilibrium. Guo *et al.*, (2013) opined that the respiratory signs in pigeons are usually absent, a fact verified in the present observations.

Although the observed number of cases were very limited, recorded signs are thought to be reasonably representative. As a general portrayal of clinical signs, affected fowls in the present study exhibited severe respiratory and enteric signs; while affected pigeons prominently showed neurological manifestations and mild enteric signs. These variations were evidently due to the different pathotypes of the strains.

The gross and histopathological lesions in the present findings conform to the description in the literature for fowls (Hamid *et al.*, 1991; Kommers *et al.*, 2003; Hines and Miller, 2012) and pigeons (El Mubarak *et al.*, 1990; Isidoro-Ayza *et al.*, 2017).

The presence of haemorrhagic lesions in the intestine has been reportedly used to distinguish between viscerotropic and neurotropic strains of the virus (Hanson *et al.*, 1973), which are characteristically present over the tips of the glands in the proventriculus, caeca, small and large intestine (Alexander and Senne, 2008b). Based on these facts, the fowl isolate were indicative of a viscerotropic virus strain. The overall nature of histopathological lesions caused by genotype XIII Newcastle disease virus were said to be similar to that of classical viscerotropic velogenic NDV with significant pathological lesions in proventriculus, intestine, trachea, lungs as well as lymphoid organs like caecal tonsils, spleen, and bursa of Fabricius (Khorajiya *et al.*, 2015). This has thus been corroborated in the present findings.

In contrast, the pigeon virus strain in the study was regarded as a neurotropic strain, even when occasional enteric lesions characterized by proventricular haemorrhages were apparent. The reaction of the nervous tissue to the virus is classically described as a non-purulent meningoencephalomyelitis (El Mubarak *et al.*, 1990; Alexander and Senne, 2008b; Isidoro-Ayza *et al.*, 2017). However, in the fowl the neurological lesions observed were not very prominent, possibly because the fowl strains were predominantly viscerotropic. Brown *et al.*, (1999) infers that the neurotropic velogenic form is more usually characterized as slower in onset, with birds in general remaining bright and alert but later becoming depressed to variable degrees, with paresis or paralysis most prominent at 5 days or later. It is speculated that the fowls die due to an overwhelming injury to the respiratory and enteric system before prominent lesions develop or become visible in the nervous tissue. On the other hand the nervous lesions in the pigeons were more overt, as were the neurological manifestations, which indicate the neurotropic predilection of the virus. It is also speculated, that the mildly pathogenic nature of the pigeon virus strains has sufficient time to generate a neurological response.

A significant finding in the nervous tissue was the presence of swollen and reactive endothelial cells of the blood vessels and the presence of vacuolation in some of the neurons, particularly of the Purkinje cells of the cerebellum. These have also been described in recent literature (Kommers *et al.*, 2003; Hines and Miller, 2012) where the authors document similar lesions from chickens infected with pigeon-origin isolates of Newcastle disease virus. In cormorants affected with Newcastle disease, endothelial hypertrophy usually was found in small blood vessels, and was characterized by large endothelial nuclei with vesiculated chromatin

protruding into the vascular lumen (Kuiken *et al.*, 1999). Other instances of endothelial hypertrophy have also been described in the brain of affected fowls (Hamid *et al.*, 1991; Ecco *et al.*, 2011). Presence of viral antigen has also been immunohistochemically documented in endothelial cells of the nervous tissue (Ecco *et al.*, 2011). Vacuolation had also been described earlier in the cerebellar white matter of spontaneously affected pigeons (Mehmood, 2017).

The presence of possible intracytoplasmic inclusions was also another significant observation. Verification with immunolocalization or electron microscopy is necessary. The presence of distinct accumulations of eosinophilic granular material present in neurons of the nucleus reticularis with strongly positive immunohistochemical reaction for viral nucleoprotein have been described by Kommers *et al.*, (2003). Rare reports for the presence of intranuclear and intracytoplasmic eosinophilic inclusion bodies in epithelial cells of oesophageal glands and in neurons of the ganglia subjacent to the adrenal gland in infected pheasants, and in hepatocytes of cuckoo doves have also been documented (Shivaprasad *et al.*, 1999). Transmission electron microscopy evidence of intracytoplasmic inclusion bodies suggestive of paramyxovirus in endothelial cells in blood vessel from the oesophagus of an NDV-infected game chicken is also on record (Crespo *et al.*, 1999).

In conclusion the present findings show that the genotypic and phenotypic variability in *Avian orthoavulavirus 1* (AOAV-1) express subtle differences in host pathology. It is therefore essential that the salient pathological changes are described and identified for assisting and differentiating in diagnosis of the disease, particularly while attributing a particular pathotype to a prevalent strain.

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