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Experimental Velogenic Newcastle Disease Can Be Very Severe and Viscerotropic in Chickens but Moderate and Neurotropic in Guinea Fowls

O. Amarachukwu Igwe[1], S. Wilfred Ezema[2], C. Didacus Eze[2] and O.A. John Okoye[2]
[1]College of Veterinary Medicine, Michael Okpara University of Agriculture, Umudike, Abia State, Nigeria
[2]Department of Veterinary Pathology and Microbiology, University of Nigeria, Nsukka, Nigeria

Abstract: Information about the pathogenesis of Newcastle disease (ND) is still limited in many avian species including some poultry birds. Four weeks old cockerels and guinea fowls were inoculated with a local Nigerian velogenic ND (VND) Virus (VNDV) strain, Kudu-113, intramuscularly. The main clinical signs in chickens were severe depression and diarrhoea while the guinea fowls showed mainly leg paralysis. Weight loss was significant in the infected birds in both species from days 3-21 post inoculation (PI). The total mortalities in the guinea fowls and chickens were 22.2 and 94.6%, respectively. The guinea fowls showed no proventricular haemorrhage, intestinal ulcers, haemorrhages and swelling of the caecal tonsil which were all prominent in the cockerels. Gross congestion of the brain was observed in the guinea fowls only. But lesions in the lymphoid organs and microscopic changes in the brain were similar in both species. The antibody response to the viral inoculation was higher in the chickens than the guinea fowls. The above observations confirm experimentally that chickens are more susceptible to VND than guinea fowls. Furthermore, the Kudu-113 infection was viscerotropic in chickens but neurotropic in guinea fowls.

Key words: Guinea fowl, chickens, pathogenesis, velogenic ND virus

INTRODUCTION

Newcastle disease (ND) is a major disease problem of poultry world-wide (Alexander, 2001; Ezema *et al*., 2009). The velogenic ND (VND) the most severe form of the disease causes fulminating outbreaks in Africa and Asia where the aetiologic pathotype is enzootic (Mathiranan *et al*., 2004; Nakamura *et al*., 2004; Oladele *et al*., 2008). VND in Europe and America where it is exotic is a grade A reportable disease. Control of outbreaks in these countries is very expensive because it is by eradication or stamping out policy which involves destruction of large populations of birds, quarantine, surveillance and payment of compensation. Outbreaks also lead to loss of export trade in poultry and poultry products. Generally the prevention of ND is by vaccination and biosecurity (Baba *et al*., 2006; Ezema *et al*., 2008; Van Boven *et al*., 2008). However, many out breaks of ND still occur in vaccinated birds (Saidu *et al*., 2004; Alexander and Senne, 2008). This could be due to the existence of highly virulent field strains of the virus which can break down vaccine immunity, use of expired and impotent vaccines and wrong vaccination procedures. Furthermore vaccination protects against clinical signs but not against lesions and some multiplication and shedding of the pathogenic field virus (Kapczynski and King, 2005; Ezema *et al*., 2009). All the ND viruses are serologically homologous but minor antigenic variations have been reported (Lim *et al*., 2003; Ibu *et al*., 2008). One of the major problems in the control and epizootiology of ND is that the virus has a very wide host range, infecting over 250 avian species including poultry, cage and wild birds (Saidu *et al*., 2004; Oladele *et al*., 2008; Ibu *et al*., 2009) The severity of the infection depends on the species, host resistance and age, virulence and dose of the virus, route of infection and presence of concurrent infections (Alexander and Senne, 2008; Ezema *et al*., 2009). This makes the manifestations of the disease often unpredictable and difficult to recognize in the field. In many host species the infection is sub clinical but these birds serve as reservoirs of infections for susceptible birds (Kommers, 2001; Saidu *et al*., 2004; Ibu *et al*., 2009). No pathognomonic lesion has been identified for ND (Hamid *et al*., 1991; Brown *et al*., 1999; Okoye *et al*., 2000) and this makes rapid diagnosis and early control measures difficult. The clinical signs, lesions and pathogenesis of ND have been described by several authors in the domestic chickens (Brown *et al*., 1999; Okoye *et al*., 2000) which are the most susceptible species to ND. Information on the other poultry species is limited. In this project we did a comparative study of experimental VND in chickens and Guinea fowls using a local Nigerian strain of the virus.

MATERIALS AND METHODS

This study was scrutinized and approved by the University Committee on Medical and Scientific Research Ethics, University of Nigeria, Nsukka.

Corresponding Author: O.A. John Okoye, Department of Veterinary Pathology and Microbiology University of Nigeria Nsukka, Nigeria

Flock history: The chickens used were cockerels which were obtained at day old from a local hatchery. But the guinea fowl keets were obtained from the Poultry Research Department of the National Veterinary Research Institute (NVRI) Vom, Nigeria. Both species were hatched the same day. The parent stocks of the cockerels were vaccinated against ND while those of the guinea fowls were not. Both groups were brooded on deep litter; feed and water were supplied *ad libitum*. The birds were not vaccinated against any disease. The principles of humane laboratory animal care were followed throughout the study.

VND virus inoculum: The virus used was VND virus (VNDV) strain, known as Kuru duck-113 (Kudu-113). The virus was isolated in Kuru Plateau State of Nigeria from an apparently healthy duck and characterized biologically by Echeonwu *et al*. (1993). The inoculum had a median embryo lethal dose (ELD_{50}) of $10^{8.46}$ per ml.

NDV challenge: At 4 weeks of age the two species of birds were each randomly divided into two groups. Group 1 cockerels were 60 birds while Group 2 contained 30. Group 1 guinea fowls contained 40 birds while Group 2 contained 35. At six weeks of age each of the cockerels and guinea fowls in groups 1 was inoculated intramuscularly (IM) with 0.2 ml of the inoculum (infected groups) The birds in groups 2 each had 0.2 ml of phosphate buffered saline (PBS) IM (uninfected groups). The four groups were reared separately in isolated pens.

Clinical signs and pathological examinations: The birds were observed twice daily for clinical signs from days 0 to 21 post inoculations (PI). Ten birds in each group were randomly selected and weighed on days 0, 3, 6, 10, 15, 21 PI. Three birds in each group were sacrificed on days 3, 6, 10, 15, 21 PI. In the infected groups the sacrificed birds were those showing clinical signs. The sacrificed and dead birds were necropsied. Samples of the spleen, bursa of Fabricius, thymus, kidney, liver, intestines and brain were collected and fixed in 10% formal saline for not less than 24 h. The tissues were processed and embedded in paraffin wax. Sections of 5 mm thick were cut and stained with haematoxylin and eosin and examined under the light microscope.

Serology: Blood samples were collected from ten birds in each group on days 0, 10, 14 and 21 PI. Serum samples were harvested and stored at -20°C until assayed. To remove non-specific inhibitors from Guinea fowl sera, the samples were adsorbed to chicken red blood cells (RBC). This was done by adding 0.025 ml of packed chicken RBC to 0.5 ml of guinea fowl sera, shaking gently and leaving for at least 30 min. The RBCs were then pelleted by centrifugation at 800 g for 5 min. The serum samples were inactivated by heating at 56°C for 30 min in water bath. The haemagglutination (HA) and haemagglutination inhibition (HI) tests were done using the microtitre methods of Beard (1989). The geometric mean titres (GMT) of the samples were calculated using the Tube Number (modified $\log_2$) and Tables provided by Villages and Purchase (1989). The antigen used for the HI test was a PBS suspension of LaSota vaccine which had 10HA unit.

Statistical analysis: Mean values and significance of differences between the mean body weights were analyzed using student t-test within groups. Significant means were separated using t-test for Equality of means and Leveue's test for Equality of variances (Chatfied, 1983). All tests were performed with a 5% level of significance.

RESULTS

Clinical signs: Clinical signs were first observed in 13% of the 60 infected chickens on day 2PI and these included ruffled feathers, depression, reduction in feed and water consumption. By day 3PI, 90% of the infected chickens were severely depressed and lethargic. Some were prostrate. There was whitish and greenish diarrhea. Some of the birds tucked their heads under droopy wings, huddled together and had hunched posture. On day 4PI, 100% of the birds were depressed. Coughing and noisy respiration with serous ocular discharges was observed in four birds. Jerking of the head was observed in one chicken while paralysis was evident in 3. By day 6PI, head tremors, wing and leg paralysis were observed in two chickens. On day 7PI head tremors and torticollis occurred in 4 chickens, paralysis in 5 and droopy wings in 5. There was full recovery by day 11PI.

The guinea fowls showed depression in 10% of the 40 infected on day 2PI. By day 4PI, 52% of them had paralysis of the legs. Some also showed jerking of the head, ataxia, recumbency and torticollis. The feathers were ruffled. There was drop in feed and water consumption and some had whitish-green diarrhea. On day 5PI the number of paralysed birds increased. Droopy wings occurred in 7 birds. Morbidity increased to 91% by day 6PI. Torticollis was seen in 9 birds. Nervous signs were prominent up to day 11PI when improvement started. Full recovery was observed on day 15PI.

Weight loss was highly significant in infected chickens and guinea fowls from days 3 through to 21PI ($p<0.05$) (Table 1).

Mortality was first observed in chickens on day 4PI when 11 birds died. Peak mortality occurred on days 5 and 6PI involving 22 and 16 birds, respectively. Only 1 chicken died on day 8PI which was the last day mortality occurred. The first mortality in the guinea fowls was recorded on day 6PI when 4 birds died and the last was

Table 1: Mean body weights of birds (g) ±SEM

	Days post infection					
DPI (Group)	0	3	6	10	15	21
CHC	455±13.84	548±13.67	643±20.09	668±19.38	790±23.33	880±31.14
CHI	475±14.43	380±8.17*	350±25.00*	375±28.87*	417±36.32*	483±22.05*
GFC	445±7.27	500±11.18	580±10.41	625±10.54	698±11.46	733±13.97
GFI	470±13.84	400±9.13*	360±12.47*	335±15.90*	403±17.66*	483±23.88*

Means with asterisks are highly significantly lower than their controls ($p<0.05$)
CHC: Chicken control
CHI: Chicken infected
GFC: Guinea fowl control
GFI: Guinea fowl infected
SEM: Standard error means to the mean values of control and infected birds

Table 2: Morbidity and Mortality in the infected groups of chickens and guinea fowl (%)

	Chickens		Guinea fowls	
Days PI	Depression	Mortality	Depression	Mortality
1	0[A]/60[B](0)	0	0/40	0
2	8/60(13.33)	0	4/40(10)	0
3	54/60(90)	0	8/40(20)	0
4	56/56(100)	11(19.64)	19/36(52)	0
5	44/45(98)	22(48.89)	30/36(83)	0
6	21/23(91)	16(69.57)	29/32(91)	4 (11.11)
7	3/7(42.86)	3(42.86)	25/28(89)	4 (12.50)
8	1/4(25)	1(25)	19/24(79)	0
9	0/3(0)	0	15/24(63)	0
10	0/3(0)	0	11/24(45)	0
11	0/3	0	5/18(28)	0
12	0/3	0	4/18(22)	0
13	0/3	0	2/18(11)	0
14	0/3	0	2/18(11)	0
15	0/3	0	1/18(6)	0
16	0/3	0	0/12	0
17	0/3	0	0/12	0
18	0/3	0	0/12	0
19	0/3	0	0/12	0
20	0/3	0	0/12	0
21	0/3	0	0/12	0
Total	56(100%)	53 (94.6%)	29(91%)	8 (22.2%)

[A]: Number positive for depression
[B]: Total number of birds per group. It is also the number of birds remaining in a group when first and subsequent mortalities were observed

4 birds on day 7PI. The total mortalities were 22.2 and 94.6% in guinea fowls and chickens respectively excluding the sacrificed birds (Table 2) the uninfected groups showed no clinical signs.

Gross lesions: Infected chickens showed congestion of the muscles of breast, thigh and legs. Haemorrhages on the mucosa of proventriculus were prominent. In some cases the haemorrhages were linear at the proventriculus-oesophagus junction (Fig. 1). Sharply demarcated ulcers were observed in the ileum (Fig. 2) while the upper parts of the intestines had haemorrhagic or catarrhal enteritis. The ulcers were evident from the mucosal and serosal surfaces. The caecal tonsils were swollen, haemorrhagic and often contained cheesy necrotic materials. Samples of the spleen were initially enlarged, mottled with dark spots on the serosal surface and later atrophic (Fig. 3). The organ later regained its normal size. The thymus was atrophic (Fig. 4) and later could no longer be detected but gradually started regaining its normal size. The bursa was initially enlarged followed by atrophy (Fig. 5). The kidneys were

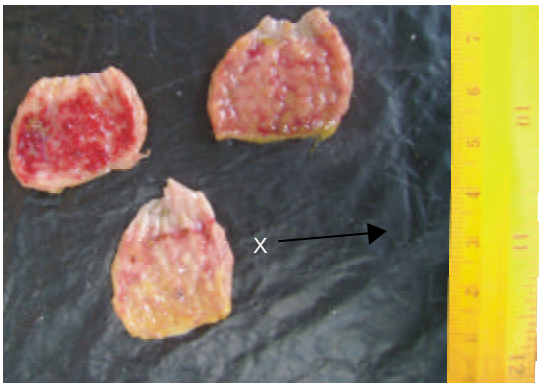

Fig. 1: Haemorrhages on the proventricular mucosa of infected chickens that died on day 4PI. Note the linear lesion at the proventriculus-oesophagus junction (X)

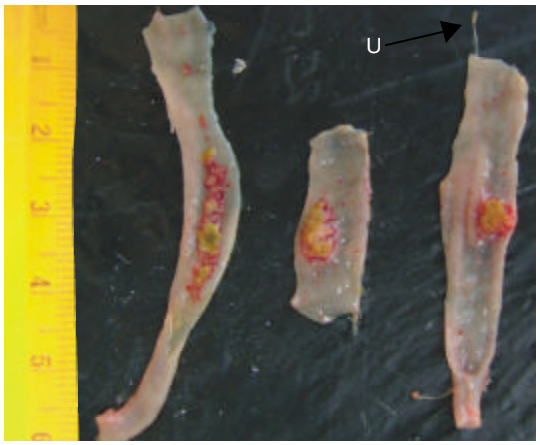

Fig. 2: Haemorrhagic ulcers (U) on the mucosa of the ileum in infected chickens that died on day 4PI

enlarged, congested and haemorrhagic while the liver showed pale or parboiled colour. The distribution and frequency of the lesions are shown in Table 3.

Infected guinea fowls showed congestion of the muscles of the breast, thigh and legs. The spleen was initially swollen mottled and later atrophic. The thymus and bursa were atrophic (Fig. 6). Grossly there was marked congestion of the blood vessels of the brain.

Table 3: Distribution and frequency of gross lesions in infected chickens

Organ	Days PI Lesion	3	4	5	6	7	8	9	14	15	21
Skeletal muscles	Congestion	0[a]/4[b]	11/11	21/21	15/15	3/3	2/2	1/1	-	-	0/3
Proventriculus	Mucosal hemorrhage	1/4	8/11	21/21	12/15	3/3	2/2	1/1	-	-	0/3
Thymus	Atrophy	1/4	2/11	19/21	12/15	1/3	0/2	0/1	-	-	3/3
	Total atrophy	0/4	0/11	0/21	0/15	0/3	2/2	1/1	-	-	0/3
Bursa	Enlargement	0/4	4/11	5/21	4/15	2/3	½	0/1	-	-	0/3
	Atrophy	1/4	2/11	9/21	6/15	1/3	½	1/1	-	-	3/3
Spleen	Mottling	0/4	9/11	15/21	9/15	0/3	0/2	0/1	-	-	0/3
	Enlargement	3/4	3/11	4/21	9/15	2/3	0/2	0/1	-	-	0/3
	Atrophy	0/4	0/11	6/21	4/15	1/3	2/2	1/1	-	-	0/3
Kidney	Congestion and enlargement	0/4	11/11	16/21	11/15	0/3	0/2	0/1	-	-	0/3
Intestine	Haemorrhagic ulcer	0/4	8/11	9/21	11/15	1/3	2/2	0/1	-	-	0/3
	Hemorrhagic or catarrhal enteritis	4/4	11/11	21/21	15/15	3/3	2/2	1/1	-	-	0/3
Caecal tonsils	Mucosal hemorrhage and enlargement	3/4	8/11	19/21	13/15	3/3	0/2	0/1	-	-	0/3
Liver	Parboiled	0/4	1/11	7/21	4/15	2/3	0/2	0/1	-	-	0/3

[a]: Number positive for lesion, [b]: Total number necropsied

Table 4: Distribution and persistence of gross lesions in infected guinea fowls

Organ	Days PI Lesion	3	4	5	6	7	8	9	10	14	15	21
Skeletal muscles	Congestion	0[a]/4[b]	-	-	4/4	4/4	-	-	6/6	1/1	0/6	0/6
Proventriculus	Mucosal hemorrhage	0/4	-	-	0/4	0/4	-	-	0/6	0/1	0/6	0/6
Thymus	Atrophy	0/4	-	-	4/4	4/4	-	-	4/6	0/1	2/6	0/6
	Total atrophy	0/4	-	-	0/4		-	-	2/6	1/1	1/6	0/6
Bursa of	Enlargement	0/4	-	-	0/4	2/4	-	-	1/6	0/1	0/6	0/6
fabricius	Atrophy	0/4	-	-	3/4		-	-	5/6	1/1	4/6	4/6
Spleen	Mottling	0/4	-	-	1/4	1/4	-	-	1/6	0/1	0/6	0/6
	Enlargement	4/4	-	-	1/4	1/4	-	-	0/6	0/1	0/6	0/6
	Atrophy	0/4	-	-	2/4	2/4	-	-	6/6	1/1	3/6	0/6
Kidney	Congestion and enlargement	0/4	-	-	3/4	3/4	-	-	0/6	0/1	0/6	0/6
Intestine	Haemorrhagic ulcers	0/4	-	-	0/4	0/4	-	-	0/6	0/1	0/6	0/6
	Catarrhal enteritis	2/4	-	-	4/4	4/4	-	-	6/6	1/1	4/6	0/6
Caecal tonsils	Mucosal hemorrhage and enlargement	0/4	-	-	0/4	0/4	-	-	0/6	0/1	0/6	0/6
Liver	Parboiled	2/4	-	-	1/4	0/4	-	-	0/6	0/1	2/6	0/6

[a]: Number positive for lesion; [b]: Total number necropsied

The gastrointestinal tract was empty in most of the infected guinea fowls with evidence of catarrhal enteritis. Both the uninfected chickens and guinea fowls showed no lesion. The distribution and persistence of the lesions are shown on Table 4.

Histopathology: The microscopic lesions in the chickens and guinea fowls were similar but the lesions were more severe in the chickens. Thymus showed hyperaemia, severe necrosis and depletion of lymphocytes. Similar lesions were observed in the spleen. In addition there was deposition of fibrin around the sheathed arterioles. By day 15PI repopulation of the spleen with lymphocytes was almost complete and there was marked increase in the number of lymphoid follicles. The bursa also had severe necrosis and depletion of the lymphocytes. Follicular epithelium was hyperplasic, hyperaemia, ballooning degeneration (Fig. 7), intra and inter follicular oedema and cystic cavities were observed. By day 7PI, the follicles were atrophic in one chicken and two guinea fowls. Sections of the brain had severe hyperaemia, degeneration and necrosis of neurons, neuronophagia, gliosis, demyelination and endotheliosis (Fig. 8, 9). There was

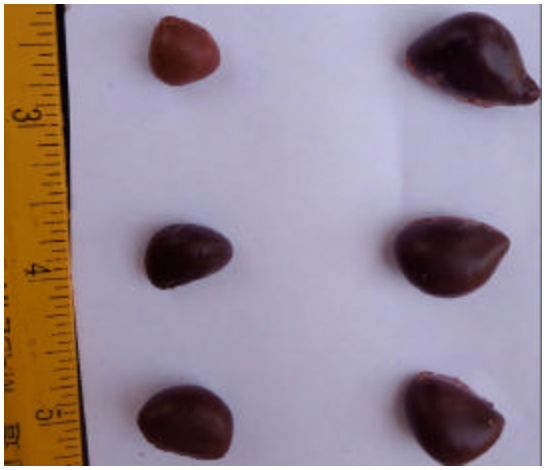

Fig. 3: Atrophy of the spleen of infected chickens that died on day 4PI

severe hyperaemia of the peritubular blood vessels in the kidney with degeneration and necrosis of the tubules. The liver in guinea fowls only showed hyperaemia of the hepatic vessels and necrosis of the

Table 5: Hemagglutination Inhibition antibody titers in infected chickens and infected guinea fowls

	Infected groups								Uninfected groups	
DPI	0		10		15		21		Day 0- 21 PI	
S/N	Ch	GF	Ch	GF	Ch	GF	Ch	GF	Ch	GF
1	0	0	256	64	4096	256	4096	256	0	0
2	0	0	256	256	1024	512	1024	1024	0	0
3	0	0	256	256	1024	512	1024	1024	0	0
4	0	0	-	128	-	256	-	512	0	0
5	0	0	-	64	-	256	-	256	0	0
6	0	0	-	256	-	512	-	1024	0	0
7	0	0	-	256	-	512	-	1024	0	0
8	0	0	-	256	-	1024	-	2048	0	0
9	0	0	-	128	-	512	-	512	0	0
10	0	0	-	128	-	512	-	512	0	0
GMT			256.	157.6	1663.5	445.7	1663.5	675.6	0	0

GMT: Geometric mean titer CH: Chickens GF: Guinea fowls

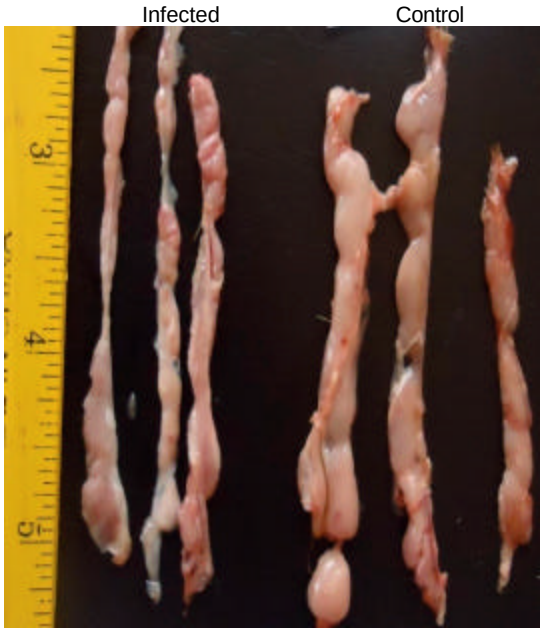

Fig. 4: Atrophy of the thymus of infected chickens that died on day 4PI

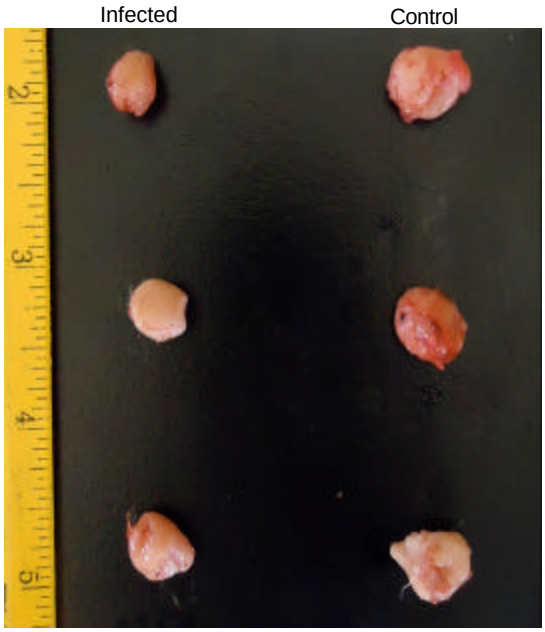

Fig. 5: Atrophy of the bursa of infected chickens that died on day 6PI

hepatocytes. Severe necrosis and destruction of the mucosa and sub mucosa was observed in the ileum of the chickens only. There was no ulcer in the guinea fowls. Organs of the uninfected birds showed no lesion.

Serology: Infection with the virus lead to seroconversion on days 10-21PI (Table 5).

DISCUSSION

Based on the severity of the clinical signs, lesions and mortalities observed, the above results confirm the general belief that chickens are more susceptible and suffer more severe form of VND than guinea fowls (Kawamura *et al*., 1987; Alexander and Senne, 2008) with the virus used in the present study. To the best of our knowledge this is the first comparative study of VND in chickens and guinea fowls and should provide accurate information on the susceptibility or resistance of the two avian species to VND. The IM route of inoculation was used to ensure that the full dose of the inoculum got into the system of each bird infected. The main clinical sign of VND in guinea fowls was paralysis of the legs which was not common in chickens. The chickens showed mainly severe depression and diarrhoea. Paralysis has also been described as the main clinical manifestation of ND in double-crested cormorants (Kuiken *et al*., 1999), ostriches (Allwright, 1996; Huchzermeyer, 1996), turkeys (Piacenti *et al*., 2006), Japanese quails (Oladele *et al*., 2008) and pheasants (Muller *et al*., 1990). The lesions in the two species were also different in some organs. The guinea fowls showed no proventricular haemorrhage, intestinal ulcers, enlargement, necrosis and haemorrhages in the caecal tonsils which were very prominent in the

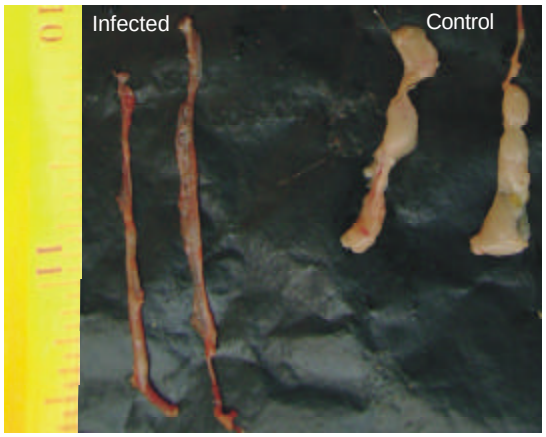

Fig. 6: Severe atrophy of the thymus of infected guinea fowls that died on day 4PI

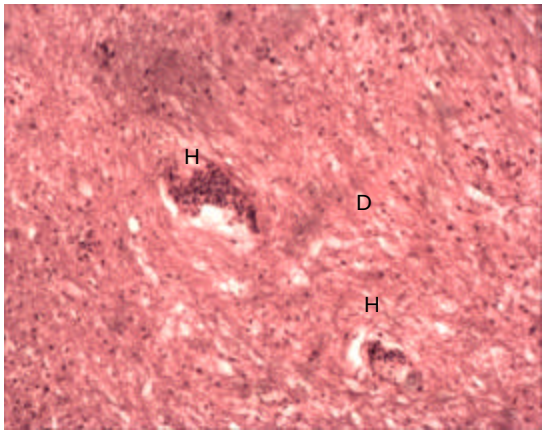

Fig. 8: Hyperaemia (H) and demyelination (D) of the cerebellum of infected guinea fowl on day 6PI. H and E X 400

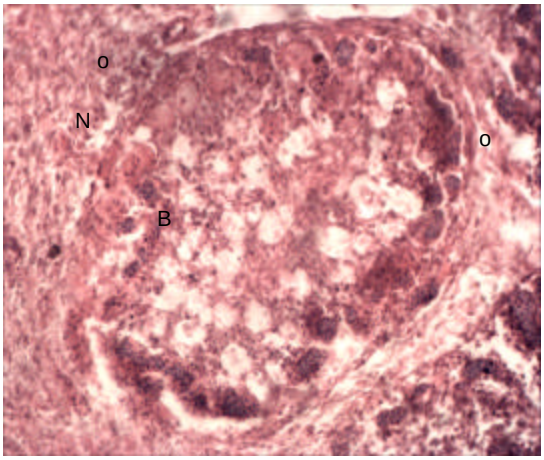

Fig. 7: Bursa of infected chicken sacrificed on day 3PI showing interfollicular oedema (O), lymphocytic depletion, necrosis, ballooning degeneration (B) hyperaemia (H) in the follicle on day 3PI. H and E X 400

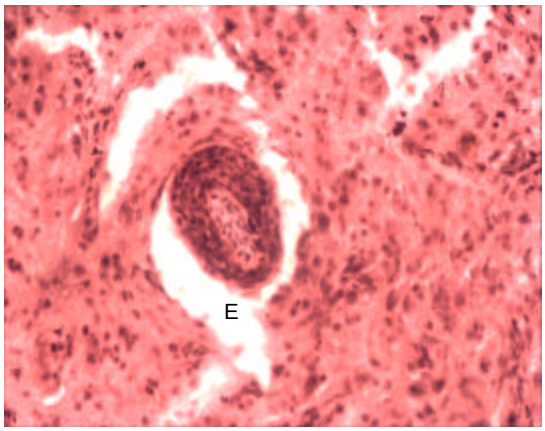

Fig. 9: Endotheliosis (E) in the cerebellum of infected guinea fowl on day 15PI. H and E X 400

chickens. The intestinal ulcers have also been reported in pheasants (Muller *et al*., 1990; Jorgensen *et al*., 1999) and geese (Wan *et al*., 2004). The gross congestion of the brain was not observed in chickens. The brain of chickens does not usually show lesions in ND (Hamid *et al*., 1991; Okoye *et al*., 2000). But the lesions in the lymphoid organs were similar, these lesions have also been observed in the double-crested cormorants (Kuiken, 1999), geese (Wan *et al*., 2004) and turkeys (Piacenti *et al*., 2006). By the classification of Alexander (2003) the NDV KUDU-113 strain used in this study is viscerotropic in chickens and neurotropic in guinea fowls. The clinical signs and lesions observed in this study in chickens are in agreement with the reports of earlier workers on VND (Hamid *et al*., 1991; Brown *et al*., 1999; Okoye *et al*., 2000; Ezema *et al*., 2008). Natural outbreaks of ND have been reported in guinea fowls by some workers (Crawford, 1931; Moine, 1950; Balarini, 1964; Durojaiye and Adene, 1988; Okaeme *et al*., 1988; Haruna *et al*., 1993). Agoha *et al*. (1992) studied experimental ND in guinea fowls using chicken strains of NDV while Mishra *et al*. (2001) studied the pathogenicity of NDV in guinea fowls using chicken and guinea fowl isolates of the virus.

They reported incubation period of 5 days, depression and leg paralysis. The clinical signs seen in the guinea fowls in this study are similar to those described by Okaeme (1988), Agoha *et al*. (1992) and Mishra *et al*. (2001). While total mortality in this study for guinea fowls was 22.2%, Agoha *et al*. (1992) observed a mortality of 100%, Haruna *et al*. (1993) 21.3% while Mishra *et al*.

(2001) recorded 8 and 52% with guinea fowl and chicken isolates respectively. Our mortality and that of Mishra *et al.* (2001) did not include sacrificed birds. In this study the incubation period of VND in guinea fowls was 2 days PI, mortalities occurred on days 6 and 7 PI only. By day 16 PI depression was no longer observed. But Mishra *et al.* (2001) reported peak mortalities on days 9 and 11PI in infected guinea fowls. Recovery from depression occurred on day 14 PI. The incubation period for chickens in our study was 2 days PI. This is in agreement with the observations of Parede and Young (1990), Ojok and Brown (1996), Galindo-Muniz *et al.* (2001) and Kommers *et al.* (2003) in infected chickens. In this study, mortalities started in chickens on day 4 PI, peaked on days 5 and 6 PI. Recovery occurred on day 9 PI. But Mishra *et al.* (2001) observed that mortalities in chickens started on day 5 PI, peaked on day 12 PI and recovery from clinical signs and death occurred on day 14 PI. Okoye *et al.* (2000) also reported that mortalities started in chickens on day 5PI, peaked on days 7 and 8 PI and by day 18 PI, only torticollis persisted in 3 surviving birds. The differences between our observations and those of some other workers could be due to the variation in the dose and virulence of the inoculum and the breeds of birds used.

The microscopic lesions observed in the bursa and spleen in this study are similar to those described by Mishra *et al.* (2001), but the gross haemorrhage they reported in the proventriculus and caecal tonsil of infected guinea fowls were not seen in this study and that of Agoha *et al.* (1992). Ezema *et al.* (2009) described ballooning degeneration in the bursa of chickens infected with VNDV strain VGF-1 and proposed that the lesion might be pathognomonic for VND. The lesion was also seen in this study in both chickens and guinea fowls. Similar lesions were shown in geese by Wan *et al.* (2004). The microscopic lesion observed in the brain in this study were also described by Agoha *et al.* (1992) and Mishra *et al.* (2001) in guinea fowls, Kuiken *et al.* (1990) in double-crested cormorants, Wan *et al.* (2004) in geese and Okoye *et al.* (2000) in chickens. The gross and microscopic lesions of VND in the bursa, thymus, spleen, proventriculus and caecal tonsil closely resemble those of infectious bursal disease (IBD) of chickens (Okoye and Uzoukwu, 1990) and highly pathogenic avian influenza (HPAI). They show the highly lymphocidal nature of the three diseases. But while the VND often produces longitudinal or linear haemorrhage on the mucosa of the proventriculus-oesophagus junction, IBD often produces similar lesion at the proventriculus-gizzard junction.

The inoculation of the chickens and guinea fowls with NDV KUDU-113 strain lead to sero conversion in chickens and the guinea fowls from days 10-21PI. The HI antibody titres in chickens were higher than those of the guinea fowls because the disease was more severe in chickens which had four times more mortalities. Saidu *et al.* (2004) in a field survey of semi-domesticated birds in Zaria area of Nigeria reported 92% positive for ND HI antibody with mean titre of 7.3 $\log_2$ in chickens while 76% was positive in guinea fowls with mean titre of 4.1 $\log_2$. In a similar survey in Jos area of Nigeria, Mai *et al.* (2004) reported 6.7% positive for ND HI antibodies in ducks and 13.6% positive in guinea fowls. The difference between the susceptibility or resistance of guinea fowls and chickens to VND could be genetic. Hassan *et al.* (2004) observed variation in the genetic resistance of four breeds of native Egyptian chickens to VND and IBD. Variation in the genetic susceptibility to important viral pathogens of poultry including Marek's disease virus, leucovirus, IBD virus and infectious bronchitis virus has been reported (Bumstead, 1998).

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