

## NEWCASTLE DISEASE IN NIGERIA

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### SUMMARY

The first documented, confirmed outbreak of ND in Nigeria occurred between December, 1952 and February, 1953. in and around Ibadan (Hill *et al.*, 1953). Subsequently, the disease appeared to be widespread in local and exotic chickens (Fatumbi and Adene, 1979; Ezokoli *et al.*, 1984; Gomwalk *et al.*, 1985; Nwosu and Okeke, 1989 ). The local chickens which are raised under the extensive system of management, where they roam freely and scavenge for food and hardly receive any prophylactic treatment or vaccine are believed to act as reservoirs and carriers of ND infection for the exotic breed (Adu *et al.*, 1986). Although ND is well studied in Nigeria it is important to present a brief review on the disease and highlight the economic importance of the disease to the poultry industry.

### EPIDEMIOLOGY

Newcastle disease (ND) had been reported from all parts of Nigeria (Hill *et al.*, 1953; Fatumbi and Adene, 1979; Ezeokoli *et al.*, 1984; Gomwalk *et al.*, 1985; Baba *et al.*, 1995). The disease was also reported to be more common during the dry harmattan (November March) (Sa'idu *et al.*, 1994a; Halle *et al.*, 1999; Abdu *et al.*, 2005a ). Cold stress has been known to worsen the outcome of ND (Abdu *et al.*, 1992). ND in Nigeria has age, and species differences (Halle *et al.*, 1999; Abdu *et al.*,

2005a ). Outbreaks of ND were reported to be more likely in farms that kept exotic birds together with local chickens and other poultry species like ducks and turkeys.( Abdu *et al.*, 2005b) . The outbreaks of ND were more common in layers than in broilers. (Abdu *et al.*, 2005a)

### SPECIE SUSCEPTIBILITY

ND was reported in both local and exotic chickens (Hill *et al.*, 1953; Fatumbi and Adene, 1979; Abdu *et al.*, 1985; Echeonwu *et al.*, 1993;

Sa'idu *et al* 1994a; Baba *et al.*, 1995; Oyeduntan and Durojaiye, 1999) The disease was also reported in the guinea fowls and turkeys (Okaeme, 1983; Ezeifeke *et al.*, 1992; Echeonwu *et al.*, 1993; Haruna *et al.*, 1993; Sa'idu *et al.*, 1994a and b; Muhammed *et al.*, 1996). A suspected case of ND was reported in the ostriches (Sa'idu *et al.*, 1999). ND virus (NDV) was isolated from apparently healthy ducks in and around Jos but there is no report of clinical ND in the ducks (Majiyagbe and Nawathe, 1981; Echeonwu *et al.*, 1993). Serological evidence of ND in the pigeons, laughing dove, mourning dove, muscovy duck, kakhi campbell ducks, geese, mallard duck, peacock, ostrich, quail, village weavers, gray-headed sparrow, red bishop, grey canary, scaly fronted weaver and bearded barbet had been reported (Ezeifeke *et al.*, 1992; Sa'idu *et al.*, 2004; Bisalla *et al.*, 2005; Ibrahim *et al.*, 2005a)

### **BREED SUSCEPTIBILITY**

The local chickens are less likely to develop clinical ND than the improved breed (intensively reared chicken) because the local breed of chickens may be infected by the mild strain of the NDV when they are scavenging for food and gradually develop immunity thus, reducing their susceptibility to severe outbreaks (Ezeokoli *et al.*, 1984.). The rural dwellers believe that there is difference in susceptibility to ND between the different breeds of local chickens (Ibrahim and Abdu, 1992), But Sa'idu *et al.*, (2005) reported that all breeds of local chickens are equally susceptible to ND. Halle *et al.*, (1999) suggested that the inability of some birds to take adequate vaccine in water during mass vaccination might have contributed to the high susceptibility of the improved breeds of chickens to ND. Breed specific morbidity rate for ND was reported to be higher for improved breeds (40 to 80 %) than for local breeds (10.6 %). This means that improved (exotic) breeds of chickens are about twice more likely to be infected with NDV than the local chickens (Halle *et al.*, 1999)

### **AGE SUSCEPTIBILITY**

Although all ages are susceptible to ND,

Chicks up to 2 weeks old with high levels of maternal antibodies may be less susceptible as implied by (Shoyinka, 1983; Ezeokoli *et al.*, 1984; Shamaki, 1989). Abdu and Garba (1989) reported that the maternal antibodies play a role in protecting chicks against ND. It was reported by Sa'idu *et al.*, (2006) that the maternal antibodies in chicks decline to a non protective level by 2 weeks of age. Halle *et al.* (1999) reported that chicks 3-4 weeks old are at high risk of suffering from ND which may be due to a decline in maternal antibody levels. It was also reported that birds within 9-10 weeks of age are more resistant to ND. This was attributed to the presence of substantial antibody titre due to ND vaccination at 6 weeks of age (Halle *et al.*, 1999). It was reported by Abdu *et al.*, (2005a) that ND affect both young and old birds.

### **ISOLATION AND CHARACTERIZATION OF ND VIRUS**

Newcastle disease virus was isolated from dead chickens and guinea fowls as well as from apparently healthy chickens, ducks and pigeons (Majiyagbe and Nawathe, 1981; Echeonwu *et al.*, 1993). All the NDV isolated from dead and apparently healthy birds by Echeonwu *et al.*, (1993) were velogenic as shown by pathogenicity indices. Isolation of velogenic NDV from apparently healthy free-roaming bird is of epizootiological importance as these may serve as reservoirs of infection to the intensively raised flocks on farms (Majiyagbe and Nawathe, 1981). Oladele *et al.* (2002) showed that the neuraminidase activity of a local isolate of NDV strain was inhibited by paranitrate oxamic acid (PNPO), salicyl oxamic acid (SOA), silver nitrate (AgNO<sub>3</sub>) and ethyl tetra acetic acid (EDTA).

### **CLINICAL SIGNS.**

The clinical signs of ND seen in chickens were:- gasping, stretching of neck, sneezing, coughing, tracheal rales, dyspnoea and opisthonus (Adene and Fatumbi, 1979 ; Okoye *et al.*, 2000). Other signs observed in ND includes drop in feed and water consumption, dropped wings, convulsion,

trembling of head and neck, backward movement and weakness or paralysis of the legs (plate 1). Abdu *et al.* (2002) described the following signs in ND; ruffled feathers, oedema of the head, cloudy eyes, conjunctivitis, severe depression, yellow and whitish diarrhoea, dehydration, emaciation and sudden death. Oladele *et al.* (2005) reported a rise in rectal temperature following infection by NDV.

The signs seen in turkeys includes tracheal rales, circling, inco-ordination, weakness or paralysis of the legs and wings, profuse yellowish green or white diarrhoea, ruffled feathers, depression and dehydration (Abdu and Sa'idu, 1990)

Clinical signs reported in the guinea fowl were circling, torticollis, inability to fly or falling while in flight, conjunctivitis, anorexia, diarrhoea, paralysis of the legs and wings, coughing, sneezing, paddling movement and sudden death (Abdu and Sa'idu, 1990; Haruna *et al.*, 1993).

## **GROSS LESIONS**

The lesion of ND reported were congestion of muscles of the breast, leg and thighs, haemorrhages were also observed on the proventricular mucosa (plate 2) while the intestines showed catarrhal or haemorrhagic enteritis. (plate 3) The jejunum and ilium often present sharply demarcated button-like haemorrhagic ulcers, these ulcers were evident from the serosal surface and usually covered by a thin greenish layer of necrotic intestinal tissue (Abdu and Sa'idu 1990; Okoye, 2000; Abdu *et al.*, 2004). Other lesions reported by Abdu and Sa'idu (1990) includes haemorrhages in the legs thigh and breast muscles, trachea, abdominal and coronary fat, airsacculitis, pale spleen and Kidneys, there is also presence of urates in the ureters, congestion of the spleen, pancreas, (plate 3) larynx,

Testis and thymus. Presence of blood in the trachea and consolidation of the lungs was also observed. Some of these lesions may be as a result of complications by some bacterial organisms like *Escherichia coli*.

In turkeys the lesions reported were haemorrhages in the gastrointestinal tract (GIT), caecal tonsils, kidneys and liver enlargement. There is also congestion of the trachea and kidneys, atrophy of the spleen and necrosis in the GIT (Abdu and Sa'idu, 1990).

In guinea fowls the lesions reported were ecchymotic haemorrhages on the tracheal mucosa, congested lungs and caecal tonsils. Catarrhal enteritis and slight enlargement and congestion of liver and spleen (Haruna *et al.*, 1993).

## **HISTOPATHOLOGY.**

Histopathologically, the following lesions were reported; mild to moderate lymphocytic necrosis and depletion of lymphocytes in the spleen. Mild lymphocytic necrosis was also observed in the Bursa of Fabricius. Submucosal oedema and ulceration of the mucosa and villi were observed in the intestines. There was also increased ulcerative haemorrhages, congestion and hyperplasia of the goblet and crypt cells. Congestion of the peritubular blood vessels, casts and pyknosis of the tubular epithelial cells and necrosis of the glomeruli and renal tubules were the histopathological lesions seen in the kidneys (plate 4). There was congestion, necrosis and mononuclear cell infiltration in the liver (plate 5). Congestion and myocarditis were observed in the heart (plate 6). There was also submeningeal oedema and mild lymphocytic infiltration in both cerebellum and cerebrum (plate 7). Vacuoles were observed in the grey matter of the cerebellum and cerebrum.

Demyelination and degeneration of the Purkinje cells were observed in the cerebellum. Perivascular cuffing with lymphocytes, oedema, congestion, gliosis and endotheliosis were observed in both organs. Necrosis of the cerebral parenchyma and markedly thickened arteries were also observed (Okoye *et al.*, 2000; Oladele 2003).

### CONTROL

In Nigeria ND is mainly controlled by vaccination. The vaccines in use are mostly live and included Hitchner B1, La Sota and komarov. ( Halle *et al.*, 1999; Abdu *et al.*, 2005a). Currently some farms are using killed oil emulsion komorov vaccines and a new viscerotropic vaccine is being tried (Adene *et al.*, 2003). The Thermostable V4 vaccine was reported to stimulate very good antibody response in local chickens ( Nwanta, 2003). However La Sota ND vaccine stimulated better immune response than the V4 ND Vaccine ( Usman, 2002; Sa'idu *et al.*, 2005)

It was reported that local chickens responded very well to vaccination with La Sota and that the vaccination reduces morbidity and mortality due to ND (Sa'idu *et al.*, 1994a; Halle *et al.*, 1999). The exotic birds also responded adequately to vaccination against ND (Shoyinka 1983). Outbreaks of ND were reported in vaccinated flocks of exotic chickens (Okoye and Shoyinka, 1983). It was suggested that the problem of ND control by vaccination in the field may be attributed to "vaccination failure" as a result of over dilution, thermal inactivation when temperature are above 50<sup>0</sup> C or "vaccine breaks" due to antigenic variation between infecting field and vaccine strains (Shamaki *et al.*, 1989)

### DIAGNOSIS.

Newcastle disease is diagnosed based on clinical signs, postmortem lesions and serological examination particularly haemagglutination (HA) and haemagglutination inhibition (HI) (Fatumbi and Adene, 1979; Haruna *et al.* 1993; Sa'idu *et al.* 1994a). Diagnosis can also be achieved by viral isolation and characterization (Majiyagbe and Nawathe, 1981; Echeonwu *et al.* 1993).

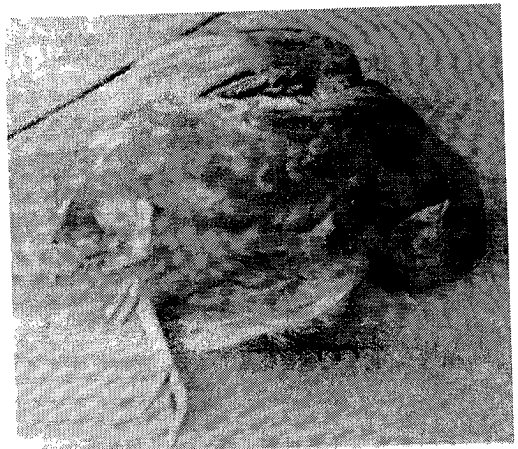


Plate 1: A layer showing paralysis of legs, wings and torticollis

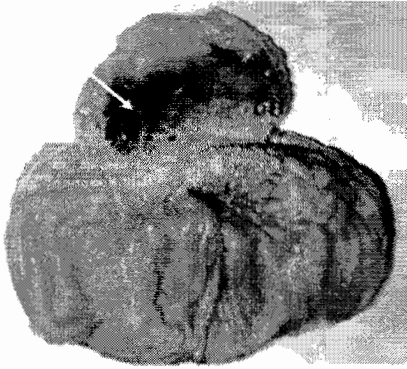


Plate 2. Haemorrhages in the cropventriculus (arrow)



Plate 3. Note, Haemorrhagic enteritis (A) and congestion of the pancreas (B)

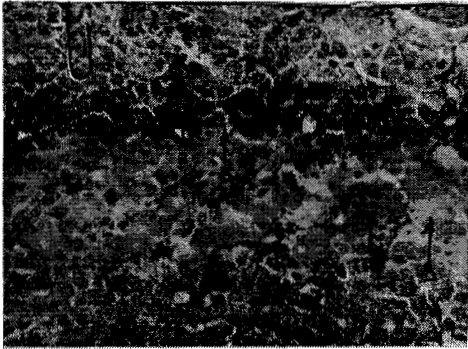


Plate 4: Kidney. Note necrosis of the glomeruli (a) and renal tubule (b). H and E x 200

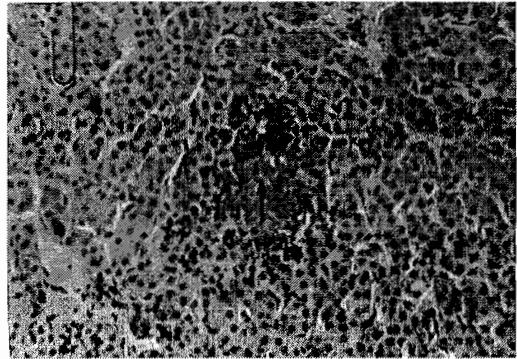


Plate 5: Liver. Note focal area of mononuclear cell infiltration (arrow). H and E x 200

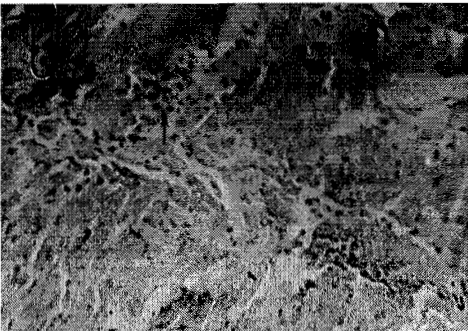


Plate 6: Heart. Note Zanker's necrosis and mononuclear cell infiltration. H and E x 200

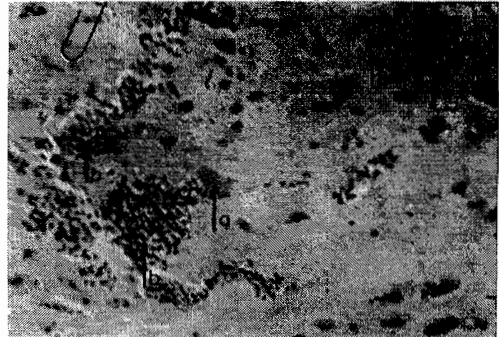


Plate 7: Brain. Note Neuronal degeneration (a) and mononuclear cell infiltration (b). H and E x 200.

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