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Susceptibility of Guinea Fowl (*Numida Meleagris Galeata*) to Infectious Bursal Disease Virus (IBDV)

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Abstract: The experiment was carried out to determine whether the guinea fowls are susceptible to Infectious Bursal Disease Virus (IBDV). A total of 60 guinea fowl keets were used for the study. They were administered the inoculum through the intraocular route. 30 guinea fowl keets served as the control group (A) while, the other 30 served as the experimentally infected group (B). The only clinical signs observed in about 10 keets were mild depression and transient loss of appetite that did not last more than 24 h. The morphometric observations also indicated that the relative weights in the two groups (A and B) showed no significant difference ($p > 0.05$) in both the Control (group A) and Infected (group B) keets. It was concluded that guinea fowls are not susceptible to IBDV but they could serve as carriers, which would spread the virus to other poultry species if reared together with them.

Key words: Susceptibility, guinea fowl, infectious bursal disease virus

INTRODUCTION

Guinea fowl is widely distributed in Africa and the husbandry practice is on the increase in Nigeria. Social acceptability studies on this species have shown promises of providing another quick source of most needed protein supplement (Ayanda and Ayeni, 1980). Under captivity the guinea fowl has shown an improvement on its egg-laying capacity from 50-100 eggs per season to about 165-185 eggs in 38 weeks (Newathe *et al.*, 1978).

This species of bird has been known to be hardier than the domestic fowl. This infers that most diseases that affect the chicken may likely not affect the guinea fowl and, if they do, the effect may be less severe in the guinea fowl. In poultry industry, the most difficult setback to the farmer is the rampant occurrence of poultry diseases, one of which is the Infectious bursal disease (gumboro disease). This is a viral disease of great economic importance.

It was diagnosed in the domestic fowl as far back as 1956 (Cosgrove, 1962). The disease has been characterized by clinical signs like prostration, severe straining during defecation, vent pecking and soiling as well as haemorrhagic diathesis. Other bird species the disease has been clinically confirmed in include Cortunix quail (Edgar and Yung Cho, 1965), English Sparrows with severe mortality (Edgar and Yung Cho, 1965).

However, experimental trials on 10 weeks old turkeys by Giron (1969) did not produce clinical signs or gross/histological changes in tissues examined. McFerrin *et al.* (1980) infected Peking duck and Village weaver (*Ploceus cucupatus*) with Infectious Bursal Disease Virus (IBDV) and they did not observe any

clinical signs. Experimental infections have been carried out on guinea fowl keets using IBDV at five zones in Nigeria (New Bussa, Ilorin, Kano, Jos and Ibadan) with the resultant manifestation of clinical disease and even death to the keets (Adewuyi, 1986). A study done at Nsukka revealed that no antibody response was obtained using agar gel diffusion precipitation test (John, 1988).

The aim of this study is to carry out experimental infection on these birds in the Northern Savannah zone and see how they will respond to IBDV.

MATERIALS AND METHODS

Experimental animals: A total of 60 guinea fowl keets, hatched in the Department of Veterinary Anatomy, Ahmadu Bello University, Zaria, were used for this study. Fertile guinea fowl eggs were obtained from the National Veterinary Research Institute (NVRI), Vom, Nigeria. These guinea fowl keets hatched were housed and fed with commercial poultry feed until four weeks before they were experimentally infected with IBD virus. Water was given *ad libitum*.

The keets were divided into two groups (A and B) of 30 keets each and separated accordingly. Group A served as the 'Control' while group B was the 'Infected' group.

Viral antigen: IBD virus from an outbreak of IBD in a local poultry farm was used in the inoculation of the keets. The inoculum was prepared by homogenizing bursal tissue of birds showing typical clinical lesions of IBD in Phosphate Buffered Saline (PBS) at a pH of 7.6 and the supernatant (inoculum) at 1:1 w/v dilution was obtained and frozen until use.

Infection: At the age of four weeks, group B was infected with the IBDV using the intraocular route. A drop of the inoculum (0.05 mL) was dropped into each widely opened eye and the drop was allowed to be absorbed before releasing each keet.

Group A had a drop of sterile water dropped into each eye in place of the inoculum. Few hours after inoculation, the keets were observed for any clinical signs at regular intervals.

The keets were randomly selected from each group (A and B) and sacrificed by euthanizing them with chloroform from day 2 post-infection (p-l). The weights of the keets were recorded accordingly. Subsequent sampling continued on days 5, 10, 13 and 15 post-infection (p-l).

Each bird was dissected open to obtain the bursal glands for gross, morphometric and histopathological observations. Normal routine laboratory procedures were used to process the tissues obtained. Sections of 5.0 μ were cut and staining was made using Haematoxylin and Eosin stains. The slides were examined under the light microscope for normal microanatomy from 'group A' and any histopathological changes from 'group B'.

Statistical analysis using Student's t-test was done on the data obtained from the live weight of the keets and the weights of bursal glands.

RESULTS

Clinical observations: Both groups of keets appeared normal and were feeding well and running around their confinement until day 2 post-infection. About 10 of the experimentally, infected (group B) keets appeared depressed and huddled together near the heat source. The feed and water intake in group B were reduced. However, these few signs were short-lived. By the 4th day, nearly all the birds were, once more, active and feeding. No mortality was recorded in both groups (A and B).

The depression and reduced feed intake were not observed with the control group A.

Gross and morphometric observations: The gross features of the bursa in both groups did not show any remarkable difference in their appearance. Each gland displayed the external evidence of 12-14 internal primary folds or plicae. These glands were oval blind sacs with short thick stalks. The mean live weight of the control keets were from 55.45 \pm 2.048-111.00 \pm 3.41g and in the infected group, the mean live weight were 55.43 \pm 2.046-109.95 \pm 2.98g (Tables 1 and 2). The mean bursal weights in the control (A) and infected group (B) were 0.061 \pm 0.002 and 0.062 \pm 0.008 g, respectively, (Table 1 and 2).

There was a difference in the mean weight that was not Significant ($p>0.05$). The relative weights in the two

Table 1: Live weight, bursa weights and relative weights of group A (Mean \pm SEM)

Control (group A) (n = 30)			
Age (Days post-hatch)	Live weight (g)	Bursa weight (g)	Relative weight (%)
30 (p-h)	55.45 \pm 2.048	0.061 \pm 0.002	0.110
33 (p-h)	58.18 \pm 1.103	0.064 \pm 0.025	0.110
38 (p-h)	63.63 \pm 2.710	0.075 \pm 0.051	0.118
41 (p-h)	80.90 \pm 3.120	0.089 \pm 0.041	0.106
43 (p-h)	111.00 \pm 3.410	0.108 \pm 0.006	0.097

Table 2: Live weight, bursa weights and relative weights of group B (Mean \pm SEM)

Infected (group B) (n = 30)			
Age (Days post-hatch)	Live weight (g)	Bursa weight (g)	Relative weight (%)
30 (p-h)	55.43 \pm 2.046	0.062 \pm 0.008	0.111
33 (p-h)	58.08 \pm 2.100	0.064 \pm 0.017	0.110
38 (p-h)	63.58 \pm 3.010	0.075 \pm 0.002	0.118
41 (p-h)	80.86 \pm 2.960	0.086 \pm 0.004	0.106
43 (p-h)	109.95 \pm 2.980	0.107 \pm 0.031	0.097

groups showed no Significant difference ($p>0.05$) also in both the control and infected groups.

The histological examination revealed only a mild distended capsule by day 13 post-infection. There was slight increase in the interfollicular connective tissue stroma. A mild increase in lymphocyte proliferation was observed in the experimental keets in both the medulla and cortical areas between days 10 and 13 post-infection. This was followed by an increase in the intensity of staining in the follicles. These slight changes disappeared by day 15 p-l.

DISCUSSION

Natural and experimental manifestations of Infectious bursal disease have been documented a lot in the domestic fowl (Okoye, 1984; Abdu, 1988; Newathe *et al.*, 1978). The disease has also been reported in other species of birds like Cortunix quill (Edgar and Yung Cho, 1965), turkeys (Giron, 1969), Peking duck and village weaver (McFerrin *et al.*, 1980; Newathe *et al.*, 1978) and English sparrows (Edgar and Yung Cho, 1965).

Experimental infections in turkey, Peking duck and village weaver showed neither clinical manifestations nor gross and histological changes. There has been conflicting reports on experimental infections in the guinea fowls. John (1988) and Hirase and Hirai (1976) found the guinea fowls to be negative to infections by IBD virus. However, Adewuyi (1986) reported cases of clinical disease and death in the guinea fowl keets that were experimentally infected.

In this study, we observed that there was no clinical manifestation of the disease in this group of birds. Despite the transient loss of appetite and depression that lasted <24 h, there were no obvious clinical signs that could be attributed to the effect of the virus in the

birds. An increase in size of the bursa which often doubles the normal size has been a prominent and consistent pathological finding of the disease in birds as early as the 3rd day of post-inoculation (Cheville, 1967). The morphometric observation in this study did not indicate any Significant difference ($p > 0.05$) in the bursal weights between the control and infected groups. The transient increase in lymphocytes could not be said to be of any major reaction to the inoculum of the IBDV to the birds. The results shown in this study are indicative that the guinea fowl is not severely affected by the virus and it could only act as a carrier of the virus.

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