The pathology of infectious bursal disease in indigenous Nigerian chickens

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INTRODUCTION

Infectious bursal disease was first reported in Nigeria by Onunkwo (11). Subsequent studies have shown that the disease in Nigeria is characterized by unusually high mortalities of up to 57 p.100 (4). The disease has affected very young chicks of 9 days old (12) and old birds of 16 to 20 weeks old (9). However, most of the published information on infectious bursal disease (IBD) of chickens in Nigeria have been obtained from exotic commercial chickens mostly imported at day-old. The disease has not been described in indigenous or local Nigerian chickens despite the fact that serological evidence of IBDV infection of the birds has been provided by Nawathe et al. (7). In this paper, the pathology of a confirmed outbreak of IBD in the indigenous Nigerian chicken is described.

MATERIALS AND METHODS

Flock history: the affected birds were 6-week-old unsexed pure local Nigerian chickens hatched and reared for experiment in the University of Nigeria Faculty of Agriculture Farm. The birds were in cages and not vaccinated against IBD despite the fact that the farm had a history of repeated outbreaks of the disease.

Clinical signs: in March 1986 the birds became sleepy with ruffled feathers and drooping wings. There were dropped in feed and water consumption and greenish diarrhoea. Prostration was commonly followed by death and mortality was 21.6 p.100.

Necropsy and histopathological changes: some of the dead birds were examined for gross pathological changes and the bursa of Fabricius, spleen, thymus, caecal tonsil and kidney were prepared for histopathology.

Virus extraction: bursae of dead birds were homogenised and 50 p.100 suspensions prepared in phosphate buffered saline (PBS). Five suspensions were tested for IBDV antigens in agar gel diffusion precipitation test (AGDT).

Serology: ten serum samples were obtained from survivors 14 days after the onset of clinical signs. The sera were inactivated at 56 °C for 30 min. and assayed for IBDV precipitins in AGDT.

Agar Gel Diffusion Precipitation Test (AGDT): the agar and method used are those already described by Okoye and Uzoukwu (9).

RESULTS

Necropsy changes: bursa was either swollen or atrophic and covered by transparent gelatinous material on the serosal surface. The mucosa was congested.
There were haemorrhages at the junction between the proventriculus and the gizzard. Spleen was motled. Kidney appeared congested and enlarged. Duodenum was haemorrhagic but the pectoral muscles were congested.

Histopathology: there were hyperplasia of the bursal epithelium, interfollicular fibroplasia and oedema with infiltration by reticular cells and macrophages (Fig. 1). The follicles were depleted of lymphocytes and contained remnants of dead lymphocytes and macrophages. Some had cystic cavities containing eosinophilic exudates and tissue debris. Changes in the spleen, thymus and caecal tonsil were similar. They were characterized by lymphocytic depletion, presence of remnants of necrotic lymphocytes, eosinophilic exudates and many macrophages (Fig. 2, 3, 4). Epithelial cells of the renal tubules and ducts showed degeneration. Some of the tubules and ducts contained eosinophilic casts. Blood vessels were congested.

Virus identification: all the 5 bursal suspensions examined for IBDV antigen in AGDT gave precipitation lines within 36 hours.

Serology: the 10 serum samples assayed for IBDV precipitins also gave positive results within 36 hours.

Fig. 1: Bursa of chicken that died of IBD showing oedema and fibroplasia in the interfollicular spaces (A) and necrotic follicles (O).

Fig. 2: Spleen of dead chicken showing lymphocytic depletion, tissue debris and macrophages in the periarteriolar lymphoid tissue (P).

Fig. 3: Thymus of dead chicken showing lymphocytic depletion, tissue debris and many macrophages in the medulla.

Fig. 4: Caecal tonsil of dead chicken showing changes similar to those of 3 and 4 above.
DISCUSSION

The Nigerian local chickens have been reported to be more resistant to diseases and nutritional stress than White Leghorns and White Rocks (2). This higher resistance could be due to the earlier growth and higher organ weight of the bursa than that of the exotic breed observed by AIRE (1). The earlier and higher organ weight of the bursa of the local chickens is likely to make them more susceptible to IBDV infection than the exotic breeds since the bursa is the target organ of IBD (6). However, the necropsy changes observed in the dead birds were less severe than those already described in field outbreaks involving exotic chickens in Nigeria (10, 11). Differences in infective viral doses and virulence of the strains could have affected the mortalities and severity of the various cases of IBD. But the histopathological changes appeared severe in all the lymphoid organs and similar to those observed in exotic birds (8). CHO, EDGAR (3) HENRY et al. (5) found no lesion in the caecal tonsil of chickens that suffered experimental IBD.

CONCLUSION

The results of this study show that the indigenous Nigerian chicken is susceptible to clinical IBD. These birds are usually neither immunized nor given any appreciable veterinary or husbandry care. It is therefore possible that they are involved in the spread of the IBDV among susceptible commercial flocks in Nigeria.
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bursae were either swollen or atrophic. Histopathological sections of the bursa of Fabricius, spleen, thymus and caecal tonsil were characterized by lymphocytic depletion and presence of remnants of necrotic lymphocytes. Suspensions of the bursa of dead chickens were positive for IBD virus antigen in agar gel diffusion precipitation test (AGDT). Sera obtained from survivors 14 days after the onset of clinical signs were also positive for IBD virus precipitins in AGDT. These observations appear to be the first description of IBD in local Nigerian chickens and confirm that they are susceptible to clinical IBD. Key words: Fowl - Indigenous Nigerian chickens - Infectious bursal disease - Nigeria.

REFERENCES