A DESCRIPTIVE STUDY OF INFECTIOUS BURSAL DISEASE EPISODES IN TWO BACKYARD CHICKEN FLOCKS IN MOROGORO – TANZANIA

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SUMMARY

Morbidity and mortality patterns as well as the gross and histological lesions which appear compatible with infectious bursal (Gumboro) disease are described in this study. The study is based on natural outbreak of the disease in two backyard chicken flocks at the Sokoine University of Agriculture in Morogoro. Both flocks showed morbidity rate of 100% and mortality rate of 38.1–40%. Clinical signs as well as gross lesions and histopathological lesions in the bursae of Fabricius were consistently the same in both flocks. The clinical signs and lesions observed resembled the classic features of infectious bursal disease. No attempts were made to isolate the virus responsible for this disease which only until very recently was not known to exist in Tanzania.

INTRODUCTION

Infectious bursal (Gumboro) disease is an important viral disease of chickens. It is extremely contagious and has a high fatality rate. It is caused by an RNA virus of unsettled classification (Gillepsie & Timoney, 1981).

Whilst there is considerable information on the clinical disease and its lesions in most of the major poultry producing areas in the world (Hofstad *et al.*, 1978), most studies of this disease in Tanzania re rather recent and casual (Anon 1989). The first serious report appear linked to the advent of heavy imports of chicks in 1988 (Kapaga *et al.*, 1989, Anon 1989).

The disease is characterised by sudden onset of high morbidity approaching 100%, usually in flocks of the age ranging between 3 and 7 weeks. The acute form has a short course and extensive destruction of lymphocytes in the bursa of Fabricius and also in other lymphoid tissue (Faragher, 1972). Severe tubular nephrosis does also occur in the kidneys. Mortality in infected susceptible chicks may be as high as 40–50% (Cosgrove 1962, Winterfield and Hitchner, 1962). Lower mortality rates are reported in infections with strains of lower virulence.

This study is based on observations in backyard chicken flocks at the Sokoine University of Agriculture, Morogoro. It describes the morbidity and mortality patterns as well as the gross and histopathological lesions which appear closely compatible with infectious bursal disease.

MATERIALS AND METHODS

Two flocks suspected to be affected with the disease were investigated with regard to morbidity and mortality patterns, management aspects and behaviour of the disease in the flocks. 60 chicken carcasses from the two different flocks were prosected and examined at the Necropsy room of the Faculty of Veterinary Medicine (FVM), Sokoine University of Agriculture (SUA), Morogoro.

The two flocks in consideration here, were of different sizes. One flock consisted of 1050 four week old chicks. The chicks were kept on rice husk litter in a newly built well ventilated and well illuminated

chicken house roofed with corrugated iron sheets. The chicks were maintained on a standard chick starter ration produced by the MIM Enterprise Morogoro. A total of 40 birds from this flock were examined by necropsy. The second flock affected consisted of 120 eight week old chicks. The chicks were also maintained on rice husk litter in a well built chicken house. A total of 20 carcasses from this flock were prosected. None of the flocks above had been vaccinated other than against New Castle Disease (NCD) and Marek's Disease (MD).

At necropsy, lesions observed in the carcasses were noted and the most remarkable ones were photographed on an ILFORD Black & White film ASA 400. Tissue samples for histopathological studies were collected from the bursa of Fabricius and spleen.

RESULTS

Morbidity features and Mortality patterns

In both flocks the affected birds appeared severely depressed inappetent and had whitish watery diarrhoea which came out with a pop sound. Feathers were ruffled and wings droopy. The flocks showed a 100% morbidity state. Many were too weak to stand or hold their heads upright.

Those that succumbed began by lying with the head tucked in under one of the wings and then went on lateral recumbency where they remained until death. Deaths began a day after the onset of the disease. Peak mortalities occurred during the 3rd and 4th day of the disease.

By the 8th day the first flock had lost 400 birds out of 1050, while the second flock had lost 48 out of 120, resulting in mortality rates of 38.1% and 40% respectively. The mortality trends in the second flock are shown in Table 1.

By the 8th day all surviving birds appeared to have recovered. Recovery was ascertained by their resumption to climb on perches and high feeders, to resume normal feeding rates as well as to start again nibbling on provided vegetables.

Table 1: The mortality trend in the second flock.

Day	Death toll	Mortality rate
1	1	0.8%
2	1	0.84%
3	18	15.25%
4	18	18%
5	10	11.36%
6	0	0

Necropsy picture

There was an amazing consistency in lesions from all of the prosected birds. All of them had petechial hemorrhages on both the pectoral and thigh muscles. Some of the hemorrhages were linear in orientation parallel to the muscle fibres (Fig.2).

The bursae of Fabricius were found swollen and oedematous in all birds (Figs. 1 & 2). Upon opening, some of the bursae contained fluid laden with whitish flakelike material resembling fibrinopurulent exudate. This was found to lie in between the plicae. The plicae were swollen with finely granular surfaces (Fig.3). Hemorrhagic lesions ranging from sparse petechiation to extensive submucosal bleeding seen in the bursae of Fabricius (Fig.3), were also seen in some birds at the junction of the proventriculus and the gizzard. Kidneys of affected birds were pale and swollen without any over-distension of the ureters. Other lesions included enlargement of the liver and increased

Figure 1: The bursa of Fabricius (arrow) is swollen as it appears in this 8-week-old pullet. Figure 2: Swollen bursa of Fabricius (large arrow) and small linear and petechial muscular hemorrhages on the pelvic limb muscles (top left corner of the picture - small arrows). Figure 3: Hemorrhagic lesions as seen on the plicae of the bursae of Fabricius (large arrows), Petechiae on bursal plicae and accumulation of puslike exudate in between the plicae (small arrows). Figure 4: Lymphocytolysis in the bursal follicles (Obj. x 40).

production of tenacious mucus in the mouth and intestines.

Histopathology

Extensive hemorrhages were present in the follicles and interfollicular tissue of the bursae of Fabricius. There was also accumulation of neutrophils in the bursal lumen. In less severely affected bursae, lesions consisted mostly of interfollicular oedema mixed with mononuclear cells and neutrophils. Lymphocytolysis was seen in the centres of the bursal follicles (Fig.4). Some lymphocytolysis was also seen in the spleen.

DISCUSSION

The rapid onset of the disease in the referred flocks, the high morbidity and mortality rate of about 40% as well as rapid recovery (7–8 days) in flocks of chicks between 4–8 weeks are features quite compatible with the behaviour of infectious bursal disease (IBD) as described by many authors (Hofstad *et al.* 1978; Gillepsie and Timoney, 1981; Gordon and Jordan, 1982).

Although isolation of the suspected virus could not be done because of the laboratory constraints existing at the FVM, Morogoro, the characteristic gross and microscopic lesions in the bursa of Fabricius and the severe intramuscular haemorrhages of the breast and thigh muscles provide adequate basis for suspecting IBD (Gillepsie and Timoney, 1981; Gordon and Jordan, 1982; and Hofstad *et al.* 1978). Paleness and swelling of the kidneys seen in the present study also conform with observations made by Randall (1985) in chickens dying from IBD. According to this author, paleness and swelling of the kidneys are caused by terminal dysfunction and are not accompanied by nephritis.

It is possible that the haemorrhages at the junction of the proventriculus and gizzard seen in some chicken indicated NCD. However, as all the chickens had been vaccinated against NCD, existence of NCD in the respective chickens could imply depression of vaccinal immunity by the IBD virus. It is known that IBD virus is able to induce immunosuppression and enhance occurrence of several infections (Otaki *et al.*, 1989). Further studies are obviously needed to establish the effect of IBD virus on NCD.

Eruptions of the seemingly IBD in Tanzania appear to be fairly recent and coinciding closely with increased importation of chicks from Europe (Kapaga *et al.*, 1989; Anon, 1989). There appears to be no record of the disease in anyone of the major animal disease laboratories in the country before 1987 as deduced from a study by Matovelo *et al.*, (1987). However, considering the severity of IBD and its impact on individual modern chicken flocks, it is likely that many other reports will follow this study.

Other major poultry diseases in Tanzania appear to be well contained through vaccines and chemoprophylaxis (Matovelo *et al.*, 1987). It is imperative, therefore, that control strategies against IBD will have to be formulated and enforced. However, further work regarding serological investigations and isolation of the IBD virus is required before such measures are taken. The authors in collaboration with other well-equipped laboratories are currently working on these aspects.

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