

Case Report

RECURRING COLISEPTICAEMIA IN BATCHES OF BIRDS IN A POULTRY FARM
IN NSUKKA, SOUTHEAST NIGERIA

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SUMMARY

Repeated outbreaks of *Escherichia coli* infection in pullets and laying birds in a poultry farm in Nsukka, southeast Nigeria are reported. The outbreaks were recorded in four batches of birds; the initial cases occurring in birds 12 – 16 weeks of age while subsequent outbreaks were in birds 28–31 weeks of age. The disease was characterized by depression, inappetence, mild cough and whitish diarrhoea; morbidity was 10% while mortality was about 5%. There was a 15% drop in egg production in laying birds. Post-mortem lesions included peritonitis, pericarditis, hydropericardium and perihepatitis. Pure cultures of *E. coli* were obtained from the organs cultured. The *E. coli* strains were sensitive to neomycin, streptomycin, gentamicin, ciprofloxacin, pefloxacin, ofloxacin and chloramphenicol but resistant to tetracycline, nalidixic acid, ampicillin and cotrimoxazole. Biosecurity measures are recommended for the control of avian colisepticaemia in Nigeria.

KEY WORDS: Outbreaks, *Escherichia coli*, pullets, laying birds.

INTRODUCTION

Escherichia coli infections could be localized or systemic and include a variety of clinical syndromes in birds such as colisepticaemia, coligranuloma, air sac disease/chronic respiratory disease (CRD), avian cellulitis, swollen head syndrome, peritonitis, salpingitis, osteomyelitis/synovitis, panophthalmitis and omphalitis/yolk sac infection (Gross, 1994; Barnes and Gross, 1997). In poultry, *E. coli* infection is typically a secondary localized or systemic disease manifested when host immune defenses have been impaired or overwhelmed (Barnes and Gross, 1997). These infections are responsible for significant economic losses to the poultry

industry (Morris and Sojka, 1985; Yerushlami *et al.*, 1990). Economic losses are usually due to high morbidity and mortality (especially in chicks) and reduction in egg production in layers. Generally, following aerosol exposure avian colisepticaemia begins as an infection of the upper respiratory tract, followed by infiltration of the blood vascular system and internal organs (Sojka and Carnaghan, 1961; Gross, 1994).

Information on cases of colisepticaemia in the poultry sector is necessary. Such information would provide basis for effective prevention/therapy of this economically important poultry disease. This paper reports on cases of

colisepticaemia in prelaying and laying birds in a poultry farm in Nsukka, Nigeria.

BATCHES OF BIRDS AND DISEASE OUTBREAKS

Batch A: Consisted of 1,120 day-old brown pullets (Shaver breed), collected from Tunes Farms/Hatchery Oshogbo, was introduced into a newly completed poultry farm.

Batch B: Comprised of 1,050 day-old brown pullets of same breed and source as batch A was also introduced into another unit of the new farm six weeks after the first batch.

Batch C: Two months after the introduction of Batch A, a third batch (Batch C) of 100 day-old broilers, collected from S and D Farms, Abeokuta, was equally introduced into the farm.

Commercial feed and water were given to the birds *ad-libitum*. Routine vaccinations and antibiotic/coccidiostat administration were carried out.

At the age of 14 weeks, there was an outbreak of disease in Batch A. The disease condition lasted for about 12 days. Neocyryl^(R), an antibiotic preparation containing neomycin, streptomycin and colistin was used in the management of the condition. The disease reoccurred at about 3 months after the onset of lay and again at 7 months into lay. Birds in batch B had an outbreak of exactly the same nature as those in Batch A at about 16 weeks of age. There were manifestations of this disease again in the Batch B birds at about 28 weeks of age. No disease outbreak was observed in birds in Batch C; however, they were sold at nine weeks of age.

The farm was left without birds for two months after which it was thoroughly

cleaned before the introduction of other batches of birds.

Batch D: Comprised of 1,800 day-old black pullets (Nera) obtained from Chi Limited, Lagos was housed at the farm.

Batches E and F: These consisted of 500 white cockerels (breed not known) and 600 black cockerels (Nera) respectively. They were both collected from Chi Limited, Lagos at day-old and brought into the farm six weeks after Batch D. Birds in these batches were brooded and sold off as brood-and-sell at 5 weeks of age.

Batches G and H: Comprised of 2,030 black pullets (Nera) and 100 broilers obtained at day-old from Avian Specialties, Ibadan, and S and D Farms, Abeokuta respectively. Both batches were also introduced into the farm four weeks after Batches E and F.

Batches D to H were subjected to similar brooding, vaccination and medication practices as was done for the first three batches.

Disease outbreaks were observed in Batches D and G at the age of 15 and 12 weeks respectively while the birds in Batches E, F and H remained apparently healthy until they were sold off.

CLINICAL SIGNS OBSERVED

The disease outbreaks were similar in clinical manifestations in all the batches. There was a sudden outbreak of a subacute disease with some of the birds showing depression, inappetence and inability to move. There was whitish diarrhoea with white pasting of the vent. Mild cough was seen in some of the birds. Mortality in outbreaks was low (less than 5%) but morbidity was up to 10%.

In laying birds, disease caused a 15% reduction in egg production but this improved as soon as the birds recovered. However, previous production levels were never reached again.

POST MORTEM LESIONS

During each outbreak 10-15 dead and moribund birds were submitted to the Department of Veterinary Pathology and Microbiology, University of Nigeria, Nsukka for patho-bacteriological examination. The post-mortem lesions seen in all the cases examined were similar. Prominent was fibrinous peritonitis characterized by fibrinous exudate in the peritoneum and mesenteries leading to serious adhesion of some of the abdominal organs. The intestines were thickened and contained cheesy exudates and broke up easily. In some of the cases, the livers were enlarged, darker in colour and with fibrinous exudates on their surfaces. Hydropericardium, fibrinous pericarditis and air sacculitis were observed in some of the cases examined at post-mortem. Breast muscles of some of the dead birds were congested.

Samples taken from the pericardium, peritoneal exudates, liver and air sacs were streaked on MacConkay and blood agar plates and incubated aerobically at 37°C for 18-24 hours. Isolates were identified based on their colonial, microscopic and biochemical characteristics (Edwards and Ewing, 1972; Cheesbrough, 2000). Antibiotic resistance profile of the isolates was determined by the agar disc diffusion method (Cheesbrough, 2000).

Profuse growth of lactose fermenting organisms in pure cultures were obtained from all the culture materials in the outbreak cases. These organisms were identified as

Escherichia coli. The *Escherichia coli* strains were sensitive to neomycin, gentamicin, streptomycin, ciprofloxacin, pefloxacin, ofloxacin and chloramphenicol but resistant to tetracycline, nalidixic acid, ampicillin and cotrimoxazole. In addition to being resistant to tetracycline, strains from Batches D and G were also resistant to cefuroxime.

DISCUSSION

The gross pathological lesions observed in the organs of affected birds are consistent with the pathologic changes associated with colisepticaemia in chicken (Cherville and Arp, 1978). Isolation of *E. coli* in pure cultures from the organs of affected chickens further confirmed that the outbreaks were as a result of colisepticaemia. Repeat occurrence of the disease outbreaks suggest re-exposure of the birds to the etiologic agent, probably as a result of persistence of the causative agent in the poultry farm. Presence of pathogenic *E. coli* serotypes as part of the intestinal flora as well as continued inhalation of these serotypes found in poultry house dust do not result in immunity (Gross, 1994).

The outbreaks reported in this study were observed in birds 12 weeks of age and above while cockerels and broilers raised in the same farm (usually sold off at 5 and 9 weeks of age respectively) were not affected. Igboke *et al.* (1996) reported an outbreak of concurrent IBD and acute colisepticaemia in 15 weeks prelayer hens in a university farm in Maiduguri, Northern Nigeria while Chah and Oboegbulem (1998) recorded outbreaks of colisepticaemia in laying flocks in Nsukka, Southern Nigeria.

The *E. coli* strains were highly sensitive to the aminoglycoside antibiotics and this may explain the effective management of the

outbreaks with drug preparations containing neomycin and streptomycin. Fluoroquinolones are a new class of antimicrobial agents that have been found to exhibit profound activity against Gram-negative bacilli (Gascia-Rodriguez *et al.*, 1995; Raemdonck *et al.*, 1992). Since the use of these fluoroquinolones in poultry may cause cross-resistance with human enteric pathogens such as *Salmonella* and *Campylobacter* spp., Blanco *et al.* (1997) strongly recommended prudent use of these antimicrobial agents in avian species. Because of variation in drug resistance patterns among the *E. coli* strains, during outbreaks of colisepticaemia the etiologic agent should be isolated and antimicrobial resistance profile determined so that the appropriate drug for the management of the condition can be selected.

As pointed out by Gross (1997), for *E. coli* infections to be effectively controlled, predisposing causes must be identified and corrected. Pierson *et al.* (1996) suggested that comparison of the serological profiles of sick and healthy flocks could aid identification of specific primary infectious agent(s) associated with the development of secondary colisepticaemia. Survashe (1996) recommended that biosecurity measures such as thorough washing and disinfection of drinkers, waterers and infected poultry houses, spraying of infected litter with 3% formalin before disposal, among others after an outbreak of Infectious bursal disease should be adopted in its control. These measures were not carried out between batches of birds in the present outbreaks. Such biosecurity measures are also recommended in the control of cases of avian colisepticaemia in Nigeria.

CONCLUSION

The present report and those of other previous workers (Igbokwe *et al.*, 1996; Chah and Oboegbulem, 1998) call for an in-depth investigation into the probable

predisposing factors to colisepticaemia in prelaying and laying chickens in Nigeria. Results of such studies will be useful in the formulation of strategies to reduce losses associated with such infections.

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