

Case Report

FOWL TYPHOID IN THREE COMMERCIAL POULTRY FARMS IN ZARIA, NIGERIA: CASE REPORTS

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SUMMARY

Fowl typhoid in three commercial poultry farms in Zaria, Nigeria, is reported. The first farm containing 30,000 battery caged shaver and Babcock layers aged 35-7 weeks, recorded 2.6% mortality. The second consisting of 2,500, Babcock layers on deep litter and aged 21 to 22 weeks, recorded 48.0% mortality. The farm also had 2,000, 9-week-old broilers with no mortality. The third farm with 2,500 Harco layers on deep litter aged 60 weeks recorded a 4.6% mortality rate. At necropsy grossly enlarged dark-greenish liver, enlarged mottled spleen and irregularly shaped haemorrhagic ovarian follicles were seen. Myocarditis, proventriculitis, hepatitis, enteritis and pneumonia were observed microscopically. *Salmonella gallinarum* was consistently isolated, in pure culture from the liver and bile. Liver and bile are good sources for the isolation of *S. gallinarum* for diagnostic purposes.

KEYWORDS: Fowl typhoid, commercial, farms

INTRODUCTION

Fowl typhoid is a septicemic, infectious and contagious disease of domestic birds caused by *S. gallinarum*. The course may be acute or chronic (Jordan and Pattison, 1999). The mortality may be moderate or high, depending largely on the virulence of the *S. gallinarum* (Shivaprasad, 1997).

The disease is endemic in Nigeria and is militating against successful operation of the poultry industry (Oboegbulem *et al.*, 1980). Though it is an important disease of poultry in Nigeria, documented reports are few (Oboegbulem *et al.*, 1980; Chima and Ogbogu, 1998). There is dearth of information on the serotypes of *Salmonella* affecting poultry in Nigeria. Out of 300 samples examined in a survey of healthy birds, only four samples of intestinal contents yielded *Salmonellae* (Falade and Ehizokhale, 1981). However, *S. gallinarum*

and *S. pullorum* were not isolated during the survey probably because diseased birds were not included (Falade and Ehizokhale, 1981).

Transmission of *S. gallinarum* is primarily due to reactor and carrier birds. These birds may infect other birds and can transmit the disease through the egg (Shivaprasad, 1997). This paper reports outbreaks of fowl typhoid in three commercial poultry farms in Zaria, Northern Nigeria.

MATERIALS AND METHODS

Flock history

In each of the three farms, flock histories, management systems and clinical signs were recorded. The first farm comprised of 30,000 Shaver and Babcock layers, aged 35-75 weeks and reared in battery cages. They had been vaccinated against Newcastle disease, Gumboro disease and

fowl pox. The disease was characterized by, 2.6% mortality rate and 20 % drop in egg production. The second farm comprised of 2,500 Babcock layers aged 21 to 22 weeks and 2,000 broilers aged 9 weeks on deep litter. Mortality rate was 48.0% in the layers and 0% in the broilers. They were vaccinated against Newcastle disease, Gumboro disease and fowl pox. The third farm consisted of 2,500 Harco layers aged 60 weeks on deep litter with no history of previous vaccination; mortality rate was 4.9% in these birds.

In all the outbreaks the consistent observed clinical signs were: purple comb and wattles, greenish-yellow diarrhoea; respiratory distress, droopiness, and/or weakness, followed by death.

Histopathological and microbiological examinations and diagnosis

Dead and clinically sick birds were randomly selected and necropsied. Gross pathological lesions were recorded. Fresh specimens were submitted for histopathological and microbiological examinations respectively. Specimens for histopathology consisted of liver, spleen, lungs, proventriculus, intestines, brain, kidneys, and heart. For histopathology tissues were fixed in 10% buffered formalin for 48hrs. The tissues were processed using automatic tissue processor (Fisher Tissuematon) as described by Culling (1974), sectioned at 5 to 6 micron, stained with hematoxylin and eosin and examined under light microscopy.

Specimens for bacteriological diagnosis consisted of swabs of the liver, gall bladder, cloaca, trachea, heart blood and shelled egg found in the oviduct at the time of necropsy. All the specimens except the

cloacal swabs, which were initially cultured in selenite F broth, were cultured on blood and MacConkey agar and then incubated at 37°C for 18 to 24hrs. Three of the pure growths of non-lactose fermenting colonies were identified biochemically using the method of Cowan (1974), by growing them in urea, triple sugar iron agar, peptone water, motility medium and for acid production in glucose, maltose and dulcitol media.

RESULTS

Gross pathological lesions observed included dark enlarged greenish friable liver, enlarged mottled spleen, irregular haemorrhagic and necrotic ovarian follicles, some of which were congested and flabby, egg yolk peritonitis, haemorrhagic epicardium, enlarged kidneys, yellowish body fat and gall bladder distended with bile. There were petechial and ecchymotic haemorrhagic areas along the ileum.

Histopathologically, tissue sections taken revealed infiltration of mononuclear cells into the myocardium, congestion of blood vessels, and infiltration of alveoli and interalveolar spaces with mononuclear cells.

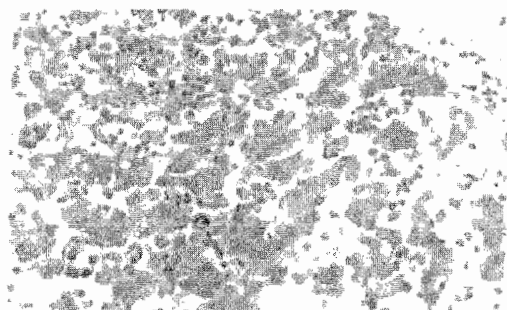


Fig. 1: Areas of necrosis of the liver infiltrated by mononuclear cells. H and E (x480)

The hepatic cells were necrotic and infiltrated by mononuclear cells (Fig. 1). Infiltration of mononuclear cells into the lamina propria, slight congestion of the tips

of villi and focal areas of necrosis in the mucosal glands were observed in the intestine (Fig. 2).

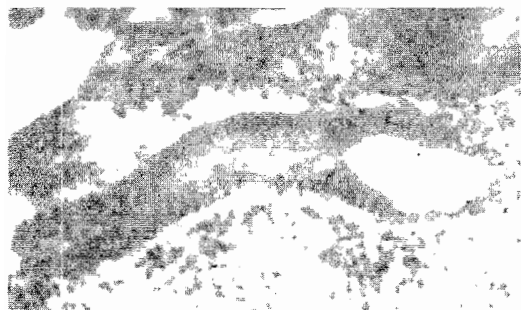


Fig. 2: Intestinal necrosis infiltrated by mononuclear cells. H and E (x480)

There was also mononuclear cell infiltration of the mucosal tips and interglandular spaces of the proventriculus. The brain, kidneys and spleen had no significant histopathologic lesions.

In each of the outbreaks, pure isolates of gram-negative organisms that were non-motile, urease negative, produced alkaline slant, acid butt and H₂S on TSI, did not produce indole, and fermented maltose and dulcitol, were obtained from all the livers and biles examined. The bacteria were sensitive to ampicillin, chloramphenicol and tetracycline and were resistant to ampiclox, augumentin, cloxacillin, erythromycin, gentamycin, penicillin, and streptomycin.

DISCUSSION

A definitive diagnosis of fowl typhoid requires isolation and identification of *S. gallinarum*. History, clinical signs and lesions are suggestive of the disease. Serology may be helpful in making a tentative diagnosis (Oboegbulem *et al.*, 1980; Shivaprasad, 1997). *S. gallinarum*

has variously been found localized in the kidneys (Kaupp and Dearstynne, 1923), ovaries (Beaudette, 1925; Beach and Davis, 1927), testicles (Gauger, 1934), and intestinal wall, liver and spleen (smith, 1955). In this report, *S. gallinarum* was found localized in the liver and bile.

Fowl typhoid, commonly produces disease in older birds and is readily transmitted from bird to bird at an age when pullorum disease is not (Hungerford, 1969). Mortality may be moderate or high depending on the virulence of the infecting *S. gallinarum* strain (Shivaprasad, 1997). In this report 21 to 75-week-old layers were involved with mortality rates of 2.6 to 48.0%. the 9-week-old broilers in which no mortality was recorded during the outbreak may be regarded as resistant. These agreed with previous reports that fowl typhoid is primarily a disease of growers and adult birds (Hungerford, 1969; Shivaprasad, 1997; Jordan and Pattison, 1999). In the present case report also Babcock appeared to be more susceptible to fowl typhoid than Harco breed.

The 2.6% mortality observed in layers in battery cages and the 4.9-48.0% mortality in layers on deep litter contrast with the report of Oboegbulem *et al.* (1980) in which there appeared to be higher mortality in layers in battery cages (18.0%) than in layers on deep litter (10.0%). This difference may be as a result of age, breed, nutritional, management and climatic differences and characteristics of exposure (Shivaprasad, 1997).

Rao *et al.* (1952) as observed in the present cases reported cyanotic instead of the anaemic comb and wattles in adult birds that is more frequently reported (Whiteman

and Bickford, 1989; Shivaprasad, 1997; Jordan and Patisson, 1999). Rao *et al.* (1952) also reported the presence of haemorrhage in the submucosa of the proventriculus. Consistent gross lesions due to fowl typhoid were marked enlargement of greenish-brown or bronze liver and congested spleen, prominent grayish necrotic patches or foci on the liver, spleen, heart muscles, kidneys and misshapen haemorrhagic ovarian follicles and peritonitis (Hungerford, 1969; Randall, 1991; Shivaprasad, 1997). The gross lesions in the liver, spleen and ovarian follicles are similar to those recorded in the present outbreaks.

Dyakov (1966) reported infarcts in the myocardium and large areas of fibrinoid necrosis and sclerosis, diffused parenchymatous hepatitis with occasional small necrotic foci sometimes infiltrated by lymphocytes. Dyakov (1966) also reported hyperplasia of reticuloendothelial system. Necrosis was observed in the heart, liver and intestines and mononuclear cell infiltration in the heart, lungs and intestines and proventriculus in the present outbreaks.

As the liver and bile consistently yielded isolates of *S. gallinarum*, in pure culture they are recommended for microbiological examination and diagnosis during suspected outbreaks of fowl typhoid.

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