

R.N. Sharma <sup>1</sup> | **An outbreak of gizzard erosion  
and ulceration in chicks in Zambia**  
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SHARMA (R.N.), PANDEY (G.S.). Début d'érosion et d'ulcération du gésier chez des poulets en Zambie. *Revue Elev. Méd. vét. Pays trop.*, 1990, 43 (3) : 301-304

L'apparition de lésions ulcératives et d'érosions sur le gésier de poulets de chair en Zambie est décrite. Environ 100 000 animaux, âgés de 2 à 9 semaines, ont été atteints et la mortalité a varié de 5 à 30 p. 100. Dans la majorité des cas, le jabot, l'oesophage et le gésier étaient envahis par une substance aqueuse dont la couleur allait du brun foncé au noir. Les lésions, principalement situées dans le gésier, étaient constituées d'érosions et d'ulcères. Un taux élevé de farine de poisson dans la ration alimentaire pourrait être l'agent causal de ces symptômes. *Mots clés* : Poulet - Gésier - Lésion - Zambie.

## INTRODUCTION

Gizzard erosion and ulceration, a recently described poultry disease is characterized by presence of blackish chocolate coloured material in the digestive tract, which runs out of the mouth if the birds are held upside down. In some countries, the disease is known as « Black Vomit » or « Vomito negro » (1, 11, 15). During the past few decades, this disease has been reported and studied in the United States of America (15), West Germany (3), Austria (5), Holland (10), Yugoslavia (4), Japan (13, 17), Peru (11), Mexico, Venezuela and Brazil (2) and Chile (15). As far as could be ascertained, there is no available literature on the subject in Africa. This paper describes an outbreak of gizzard erosion and ulceration in chickens in Zambia.

## MATERIALS AND METHODS

The disease broke out mainly in broilers during April-May 1985. A total of 47 poultry farms were investigated to study broilers affected by the disease.

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## Clinical and epidemiological examinations

The birds were clinically examined in the affected farms. Data regarding the type and source of feed, schedule of feeding, origin of chickens, their age and vaccination covering were obtained from each farm and critically analysed.

## Pathological examination

*Postmortem* examination of the spontaneously dead or clinically sick chickens after sacrifice was carried out. The gross lesions in various tissues were recorded. Pieces of oesophagus, proventriculus, gizzard, intestine, liver, kidney, pancreas, heart and spleen were collected in 10 % formol saline. After processing through paraffin embedding, tissue sections were stained with hematoxylin and eosin and examined for microscopic lesions.

## Bacteriological and toxicological examination

Twenty-one feed samples collected from the affected farms as well as liver, spleen, and gizzard with its content from 64 necropsied chicks were cultured for bacterial and fungal isolations. Paired feed samples were also analysed at the toxicological laboratory of the National Council for Scientific Research, in Lusaka, for the presence of aflatoxins.

## RESULTS

### Outline of field outbreak

Among 47 farms, 2 had 3-week old layer chicks and 45 farms had multi-aged broiler growers from 1 to 9 weeks of age. Chick numbers on the farms ranged between 1000 and 9000. Chicks were obtained mainly

from the two hatcheries of the country. Broilers were fed on broiler starter finisher mash. Unfortunately because of feed shortage the layer chicks involved in the outbreak were fed on broiler starter mash. Feed for all affected farms came from the same milling company. According to the mill records a few batches of broiler starter and finisher mash contained over 12% fish meal. In the affected farms feeding was performed with particular batches of feed 7-10 days before the onset of the symptoms. All farms had deep litter systems and feed and water were given *ad. lib.* Chicks got vaccination covering for Newcastle disease, infectious bronchitis and Gumboro disease according to the same schedule as in Zambia.

### Clinical symptoms

No clinical signs were seen in chicks younger than 8-day old. Signs of the disease were observed in chicks between the age of 9 and 60 days. Clinical signs included diarrhoea, listlessness, dehydration, weakness of legs and anaemia. In a majority of chicks, the crop was seen engorged with black material, which ran out of the mouth when birds were picked up by the legs. Most of the birds died within 4-5 days after the occurrence of symptoms. There was an appreciable weight loss in survivors.

Mortality in broiler farms ranged between 5-10% in 2-3 week old chicks, rose to 25-30% in 4-7 week old and declined again to 6-15% in 8-9 week old animals. In layer chicks mortality was below 5% in both affected farms.



Photo 1 : Gizzard showing ulcers and haemorrhages.

### Pathology

*Postmortem* examination revealed the presence of dark brown to black watery material in the digestive tract, *i.e.* mainly crop, oesophagus and gizzard. In a number of birds the proventriculus were distended and the musculature had become flaccid. The lesions were more constant in the gizzard where the early lesions included discolouration of the mucosa to dark brown or sometimes to yellow white. In some cases, the gizzard lining was rough and thickened giving a bark-like appearance.

In advanced cases, multiple cleft-like erosions were seen especially in the depths of folds. In some chicks, deep irregular ulcers, varying from a few mm to 2 cm in diameter, were discovered mixed with areas of haemorrhages (photo 1). The gizzard content in such cases was tarryblack and attached to the damaged mucosa. In approximately 5% dead chicks, ulcers had perforated through the gizzard muscle and black tar-like material had accumulated in the abdominal cavity, producing peritonitis. Changes in other tissues included enteritis, discolouration and moderate swelling of spleen, liver and kidneys.

Microscopic changes of the gizzard in early lesions, consisted in a widening and loosening of the keratinized layer with a poor staining. At places, small erosions were present in the lining. The glandular cells showed a pronounced swelling. In advanced cases, the gizzard lining was thickened and loose. The cells in the upper part of the glandular layer were swollen and a thin secreted substance with many desquamated cell debris was retained in the glandular ducts. Erosion and ulcers, which involved only the lining layer in early lesions, had extended into glandular and sometimes to muscular layers. Large areas of heterophilic infiltration and haemorrhages were seen near the ulcers.

Microscopic lesions in the proventriculus were confirmed to be a catarrhal inflammation. Intestines showed varying degrees of enteritis, sometimes leading to necrosis of the mucosa. Other tissues did not reveal appreciable microscopic changes.

### Cultural and toxicological results

All samples of feed and tissues proved to be negative for all pathogenic bacteriae and fungi. Feed samples were found to be negative for aflatoxins.

### DISCUSSION

The clinical signs, gross and microscopic lesions observed in the present outbreak of gizzard erosion

and ulceration were similar to those described by earlier workers (2, 8, 9, 11, 13, 15).

The clinical signs appeared after 8-10 days of feeding a particular batch feed, and mortality was maximum in 4-7 week old chicks. These observations are in keeping with the findings of COVER and PAREDES (2), JOHNSON and PINEDO (11), BAKKER (1), RUMSEY (16) and MONTES *et al.* (15). The mechanism of age related resistance is not well understood.

Based on the findings of field outbreaks and experimental studies, other authors (6, 10, 11, 12, 13, 14, 15, 17) previously indicated that the disease is caused by feeding diets containing over 12% of certain fish meals. The exact toxic factors in the fish meal have not yet been recognized. HORAGUCHI *et al.* (7), in an experimental study, produced gizzard erosion and ulceration in broiler chicks fed fish meal heated at 130 °C for 5 hours. Fish meal did not cause lesions without heat treatment. They concluded that this pathology was caused by feeding a high level of some fish meal by-products. ITAKURA *et al.* (8) in a separate study, supported the findings of HORAGUCHI *et al.* (7) and indicated that treatment of fish meal in the processing plants in some way alters the product nature. It has also been observed that not all the

batches of fish meal from a given source caused the disease (10, 14).

In the present outbreak, the diet contained 12-13% fish meal, which seems to be the level indicated as toxic by previous workers. The feed at the affected farms was changed by another diet containing 8% fish meal, and obtained from a separate source. Mortality subsided within 4-5 days. However, the affected birds did not show normal growth and remained stunted.

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SHARMA (R.N.), PANDEY (G.S.). An outbreak of gizzard erosion and ulceration in chicks in Zambia. *Revue Élev. Méd. vét. Pays trop.*, 1990, 43 (3) : 301-304

An outbreak of gizzard erosion and ulceration in broilers in Zambia is described. Approximately 100.000 chickens in the age group of 2-9 weeks were affected. Mortality ranged from 5 to 30%. In a majority of cases, crops, oesophaguses and gizzards were engorged with dark brown to black watery material. Lesions were mainly in the gizzard comprising erosions and ulcers. High levels of fish meal in the ration is indicated as a causative factor. *Key words* : Chick - Gizzard - Lesion - Zambia.

SHARMA (R.N.), PANDEY (G.S.). Principio de erosión y de ulceración de la molleja en pollos en Zambia. *Revue Élev. Méd. vét. Pays trop.*, 1990, 43 (3) : 301-304

Se describe la aparición de lesiones ulcerativas y de erosiones en la molleja de pollos de cría en Zambia. Se la observó en unos 100 000 animales, de 2 a 9 semanas de edad; la mortalidad varió de 5 a 30 p. 100. En la mayoría de los casos, una sustancia acuosa de color pardo oscuro a negro invadía el esófago y la molleja. Se constituían las lesiones, encontradas principalmente en la molleja, de erosiones y de úlceras. Una cantidad elevada de harina de pescado en la ración alimenticia podría ser la causa de dichos síntomas. *Palabras claves* : Pollo - Molleja - Lesión - Zambia.

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