

Histopathology of *Ascaridia galli* Infection on the Liver, Lungs, Intestines, Heart, and Kidneys of Experimentally Infected Domestic Pigeons (*C. l. domestica*) in Zaria, Nigeria.

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ABSTRACT

A study on the histopathology of *Ascaridia galli* infection on the liver, lungs, intestines, heart, and kidneys of experimentally infected domestic pigeons was carried out at the postgraduate laboratory of the department of Biological Sciences, Ahmadu Bello University, Zaria, Nigeria. Two groups (A and B) made up of 30 birds each, were used. Birds in group A were each infected with a 700 dose of infective eggs of *A. galli* while birds in group B served as controls.

Clinical signs observed in the infected birds were blood-tinged diarrhea, loss of appetite, birds looking dropsy, head nodding downwards, ruffled feathers, shivering and emaciation. The experiment was terminated 12 weeks after infection, and at necropsy, a total of 87 young adult worms were recovered from 14 birds in the infected group and none from the controls. Histopathologic analyses of the liver, lungs, intestines, heart, and kidneys of some of the infected birds were carried out to show histopathological effects. The implications of these findings are discussed.

(Keywords: *A. galli*, histopathology, pigeons, infection, parasites, poultry, fowl, ascariasis)

INTRODUCTION

Ascaridia galli is a common parasite of poultry and has been reported in chicken, turkey, guinea fowl, pigeons, duck, and goose (Ruff and Norton, 1997). It has been reported as a common parasite of pigeons and doves in Zaria (Abdullahi *et al.*, 1992; Oniye *et al.*, 2000; Audu *et al.*, 2004; Gadzama *et al.*, 2005).

The life cycle is simple and direct, and infective eggs containing the L₂ larvae, hatch in either the proventriculus or the duodenum of the susceptible host. The prepatent period is about five to six weeks.

Ascariasis is an intricate problem to poultry breeders, and so it could be to pigeon breeders and fanciers. It is one of the major causes for the reduction in egg production, reduced growth rate in broilers and consequently responsible for economic losses to the poultry industry. Perusal of the available literature indicated, very meager published information exists on the histopathological effects of *A. galli* infection in pigeons.

The purpose of this study was thus to evaluate the histopathological effects of *A. galli* infection on experimentally infected Domestic pigeon (*C. l. domestica*) in Zaria, Nigeria. This was based on histopathologic analyses of the liver, lungs, intestines, heart and kidneys, affected in the course of infection.

MATERIALS METHODS

Procurement and Acclimatization of Birds

A total of 60 pigeons comprising of 30 males and 30 females, bought from Sabo and Samaru markets in Zaria, Nigeria were used for the experiment. The birds were housed in the postgraduate animal laboratory of the department of Biological Sciences, Ahmadu Bello University, Zaria, Nigeria. The birds were acclimatized for a period of three weeks prior to the commencement of the experiment. During this

period, the birds were checked and treated for various parasites, to certify them parasite-free. At the end of the acclimatization period, the birds were divided into two groups of 30 birds each, consisting of 15 males and 15 females.

Group A comprised of infected *C. l. domestica* while group B comprised of non-infected *C. l. domestica* (controls). The birds in each group were tagged with numbers for proper identification during data collection.

The birds were fed *ad libitum* and via cocktail or cafeteria style, with guinea corn and millet, red maize and groundnut as sources of protein. Vitalites were added to drinking water as recommended to cater for vitamins and mineral salts. Water and feed were provided in drinking and feeding troughs. The cages were fitted with dropping boards that were regularly emptied.

Production of Infective Eggs of *Ascaridia galli*

Eggs used for infection were obtained from live adult females of *A. galli* collected from pigeons slaughtered at Sabo, Samaru and Tudun wada markets all in Zaria, Nigeria. The worms were collected in specimen bottles containing 0.9% physiological saline and taken to the laboratory.

In the laboratory, the worms were crushed using a mortar and pestle in distilled water to recover the eggs from uteri. The crushed worms were then filtered out using a mesh of 0.01 mesh size into a beaker. The filtrate was then allowed to stand for about an hour after which the supernatant was decanted. The sediments were then washed with 0.5 M sodium hydroxide solution into a beaker and agitated gently for 30 minutes in order to dissolve the sticky albuminous layer of eggs and allowed for uniform sampling (Fairbairn, 1970; Hansen *et al.*, 1954).

This was then placed in centrifuge tubes and centrifuged at 1500 rpm for 3 minutes to recover the eggs. The recovered eggs were then washed three times in distilled water and also three times in embryonating fluid which was a solution of 0.05 M sulfuric acid.

The eggs collected were suspended in embryonating fluid and placed in plastic troughs. These were then left to stand for 12 days in the laboratory at 30°C.

Embryonating fluid was periodically added to the egg cultures to avoid drying. Embryonated eggs were stored at room temperature for two weeks before infection of the birds.

Bird Infection

The birds were dosed by taking equal amounts of agitated egg suspension with 5 ml syringe and injecting directly into the crop, using 20 G x 1.5 inch needles. The birds in group A (infected birds) each received 0.75 ml of egg suspension containing 700 viable eggs.

The birds in group B (non-infected birds) serving as controls, were each given 0.75 ml of egg-free suspension fluid (Sucrose solution).

Fecal sample examination was carried out using simple floating technique, from the second week after infection, until infection was ascertained by detection of ascarid eggs in the feces of infected birds. The experiment was terminated 12 weeks after infection.

Histopathology

Tissue sections of liver, lungs, intestines, heart, and kidneys from the freshly slaughtered birds were immediately taken to the histopathology laboratory of the department of Veterinary Pathology and Microbiology, Veterinary Teaching Hospital, Faculty of Veterinary Medicine, Ahmadu Bello University, Zaria, Nigeria, for histopathologic analyses.

RESULTS

Clinical Signs

Blood-tinged diarrhea, loss of appetite, increased thirst, birds looking drowsy head nodding downwards, puffing or ruffled feathers and shivering emaciated and dirty cloacal region, were some of the clinical signs observed among the infected birds.

Worm Recovery

At termination of the experiment, a total of 87 young adult worms were recovered from 14 birds in the infected group and none from the non-

infected group, at necropsy. The highest number of worms recovered from a single bird was 12 and the lowest was 2.

Histopathology Report

The report showed that the liver of infected birds had fatty degeneration and areas of coagulation necrosis of the hepatic cells most predominantly at the portal areas. There were mononuclear and polymorphonuclear cellular infiltrations in the necrotized areas. The liver had congested blood vessels and congested sinusoids (Plate 1).

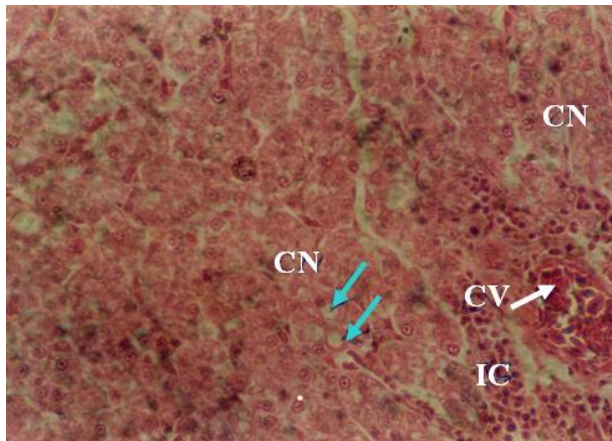


Plate 1: Photomicrograph of a Section of Liver from Pigeon.

Note: Coagulation Necrosis (CN) of the hepatic cells, fatty degeneration (arrow heads), Inflammatory Cells (IC), and Congested Central Vein (CV). H&E Stain. X400

The lungs of the infected pigeons had hemorrhagic areas, congested blood vessels and haemosiderosis. There was mononuclear and polymorphonuclear cellular infiltration at the peribronchiolar and interalveolar septae which extended and filled some alveoli (Plate 2).

The infected pigeons had necrosis of the intestines that involved the villi, intestinal glands and the muscularis mucosae of the intestines. There were mononuclear and polymorphonuclear cells in the necrotized areas (Plate 3).

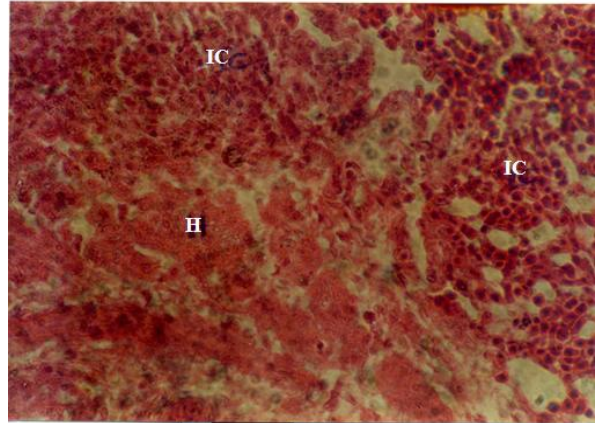


Plate 2: Photomicrograph of a Section of Lungs from Pigeon.

Note: Hemorrhage (H) and the Inflammatory Cells (IC) in the Lungs. H&E Stain. X400

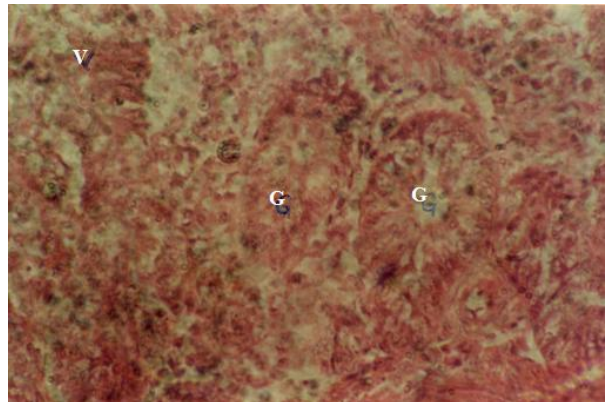


Plate 3: Photomicrograph of a Section of Intestines from Pigeon

Note: The necrosis of the Intestinal Villus (V) and Intestinal Glands (G). H&E Stain. X400

The heart had focal areas of necrosis of the myocardial cells and few mononuclear and polymorphonuclear cells in the necrotized areas (Plate 4).

The kidneys had renal tubular necrosis infiltrated by few mononuclear and polymorphonuclear cells (Plate 5).

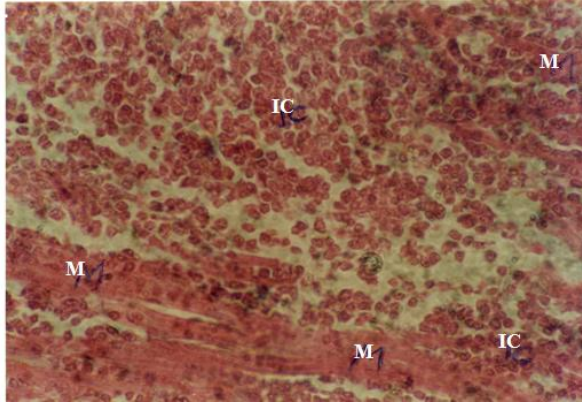


Plate 5: Photomicrograph of a Section of Heart from an Infected Pigeon, Showing the Necrosis of the Myocardial Cells (M) and the inflammatory Cellular Infiltration (IC). H&E Stain. X1000

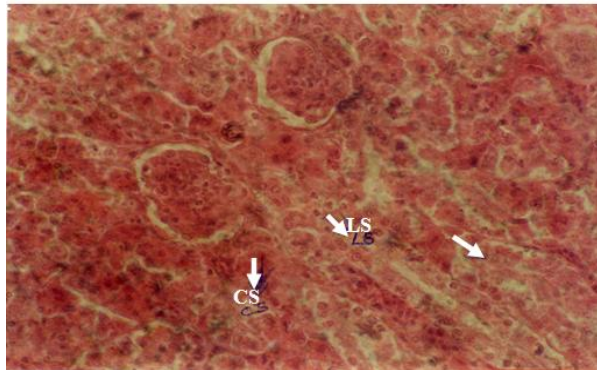


Plate 5: Photomicrograph of a Section of Kidneys from an Infected Pigeon, Showing the Renal Tubular Necrosis (Arrow Heads). H&E Stain. X400

DISCUSSION AND CONCLUSIONS

The clinical signs observed in this study, have been reported by Ikeme (1971a); Soulsby (1982); and Reid and Carmon (1958). Ntekim (1983) observed delay in the commencement of egg-laying and laying inefficiency in infected chickens.

The recovery of young adult worms, in this study is in accordance with previous reports (Reid and Carmon, 1957; Ikeme, 1971b; Ntekim, 1983). The low number of worms (87) recovered from the infected birds agrees with the observation of Roberts (1937); Sadun (1948); Reid and Carmon (1957) and Ikeme (1971b), who noted that despite large number of eggs fed per bird, only a few

worms were recovered, most of which were very small in size.

The histopathological effects particularly hemorrhagic lesions observed in the liver, lungs and intestines, may be linked to the migration of the larvae during the tissue phase of the life cycle. It has been reported by Ikeme (1971a) that adult worms when present in large numbers, migrated up and down the intestinal lumen. The adults also aggregate in the lower half of the intestine where they cause intestinal obstruction and death of the affected pigeons. In severe infections, intestinal blockage occurred and chickens infected with a large number of ascarids, suffered from loss of blood, reduced blood sugar content, increased urates, shrunken thymus glands, retarded growth, and greatly increased mortality.

Soulsby (1982) reported that in many cases, the intestinal mucosa also reveals inflammatory lesions and focal hemorrhages caused by the burrowing of parasites. This confirms the results of the present study.

It is likely from the present study that *Asaridia galli* infection could have some histopathological effects on the heart and kidneys, though no such reports exist to the best of our knowledge. These being vital organs of the body, such effects on them, could lead to high morbidity or mortality, or could lead to secondary infections or even complicate the courses of other infections or diseases in domestic pigeons. It is hereby recommended that further research be conducted to ascertain any histopathological effects of *Ascaridia galli* infection on the heart and kidneys, in support of the present study.

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