Guinea Fowl Mortality Associated with *Ascaridia numidae* Infection

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Abstract In an organized poultry farm 8-12 weeks old grower flocks of pearl variety guinea fowls have shown symptoms like anorexia, diarrhea, lethargy and emaciation. There was also 3.5 % mortality in the flock. Postmortem revealed the highly inflamed mucosa severely studded with ascarid larvae at the jejunum and ileum region. Few adult parasites were present in the lumen and were identified as *Ascaridia numidae* based on their microscopic morphology. Pooled droppings from pens were examined for EPG. Histopathology revealed numerous larvae in the lumen, mucosa and submousa of the intestine. Treatment with piperazine adipate significantly reduced the mortality to 0.8 %. EPG was significantly reduced after treatment. All these findings indicated that the cause of morbidity and mortality in pearl variety guinea fowls was due to *A. numidae* infection.

Keywords Guinea Fowl; Ascaridia Numidae; Morbidity; Mortality; EPG; Histopathology

1. Introduction

The Guinea fowl (*Numida meleagris*) is one of the important and interesting gallinaceous birds being farmed for centuries in the Indian subcontinent. Because of its low input requirements and better forage utilization capacity, guinea fowls hold a unique status for alternate poultry production for the rural unemployed youths and women. It is reared for food, game, fancy and also as a pet. The guinea fowls are highly susceptible for gastro intestinal helminth parasites because of the rearing methods and feeding habits. Helminthiasis is considered as one of the most significant constraints on poultry production in tropical climatic conditions of India which are favourable for faster propagation and development of the larval stages of helminth parasites (Matta and Ahluwalia, 1981; Kulkarni et al., 2001). Ascarid worms are one of the more common parasites infecting poultry such as guinea fowls, chickens and turkeys. The eggs of these worms were excreted in the droppings and the eggs can survive in the environment for a couple of years. The susceptible birds get infection by ingestion of these eggs. These worms have significant economic impact on the poultry farming due to the reduced feed conversion efficiency, decreased growth rate and mortality in the worm infected birds (Katoch et al., 2012).
Different helminth parasites including *Ascaridia numidae* infecting guinea fowls have been reported from South Africa (Junker et al., 2008) and Ghana (Hodasi et al., 1976). In Turkey *A. numidae* infection has been reported from rock partridges (Avcioglu et al., 2008). It is found in the lumen of small intestine or caecum (Saif et al., 2003) of guinea fowl. *Ascaridia numidae* are much smaller than *A. galli* which is a very common parasite of poultry. *Ascaridia numidae* infection in guinea fowl have been reported in northern parts of India (Gupta and Acharya, 1971; Matta and Ahluwalia, 1979) but it has not been reported from southern peninsula of India. This report describes mortality in 8-12 weeks old pearl variety guinea fowl flock in Institute of Poultry Production and Management, TANUVAS due to *A. numidae* infection.

### 2. Materials and Methods

#### 2.1. History

Pearl variety guinea fowls of 8-12 weeks grower age group had shown symptoms like anorexia, diarrhea, lethargy and emaciation. During this period, there were 74 mortalities per week in this flock which was 3.5% of the total stock. Morbidity was around 50% of the flock. These guinea fowls were fed with commercial grower feed. They were grown in eight pens with 60-70 birds/pen.

#### 2.2. Faecal Examination

Pooled droppings from eight individual pens were collected from the farm to rule out gastro intestinal parasitism. These samples were examined for Egg per Gram of feces using Modified Mc Master technique for greater sensitivity. Briefly four gram of droppings was suspended with 26 ml of saturated salt solution and mixed thoroughly. This was loaded in to a three chambered Mc Master slide and kept undisturbed for 2 minutes for the eggs to float to the top layer. Total number of eggs present under grid lines were counted and multiplied by 25 to get the EPG.

#### 2.3. Necropsy

Necropsy was conducted on three guinea fowl carcasses received from the farm as per standard procedure.

#### 2.4. Parasites Identification

Parasite samples preserved in 95% ethanol were treated with Lactophenol and examined under trinocular microscope for morphological characteristics.

#### 2.5. Histopathology

Tissues fixed in 10% formalin were embedded in paraffin. Using microtome, thin sections were made after standard processing. These sections were stained with haematoxylin and eosin and examined under trinocular microscope with image capture software.

#### 2.6. Treatment

Birds were treated with Albendazole @ 10mg/kg body weight in water (40 litres / 1000 birds). But mortality was not controlled after treatment with Albendazole then birds were treated with Piperazine adipate @ 300mg/kg body weight. Second dose was administered 14 days after the first treatment.
3. Results

3.1. Fecal Examination

EPG ranged from 1100-1500. Eggs of *A. numidae* are oval in shape with smooth shells and unsegmented. They measured 61 µm in length and 32 µm in width.

3.2. Necropsy

Carcasses were emaciated and poor in condition. There was paleness of visible mucous membrane. Liver showed multifocal pale areas. Intestinal contents were scanty as there was anorexia. Mucosa of intestine was heavily studded with ascarid larvae (Figure 1) both in the jejunum and ileum region in all the three birds. There were few adult parasites in the ileum region. No significant lesions were noticed in other organs.

![Intestinal Mucosa Heavily Studded with Ascarida numidae](image)

*Figure 1: Intestinal Mucosa Heavily Studded with Ascarida numidae*

3.3. Parasite Identification

Biometry of male and female parasites was 21 mm length and 0.9 mm width. And 29 mm in length and 1.3 mm in width respectively. Oesophagus length of male nematode was 1.4 mm and female was 1.7 mm. In male nematode there was a single papilla on the pre anal sucker (Figure 2). It is an important morphological feature of *A. numidae* which differentiates it from *A. galli* beside the difference in size.
3.4. Histopathology

There was catarrhal inflammation and diffuse lymphocytic infiltration in the mucosa and submucosa. Numerous second stage larvae were present in the mucosa and submucosa (Figure 3 and 4). There was also hyperplasia of goblet cells in the mucosa and submucosa. Clusters of bacilli were observed in the cellular debris of the lumen. In liver, there was periportal hepatitis and diffuse vacuolar degenerative charges in the hepatocytes.
Figure 3: Numerous Second Stage Larvae were Present in the Mucosa and Submucosa

Figure 4: Numerous Second Stage Larvae with Hyperplasia of Goblet Cells in the Mucosa and Submucosa
3.5. Treatment

Initial treatment with Albendazole @ 10 mg/kg body weight in water (40 litres / 1000 birds) did not control mortality and then the birds were treated with Piperazine adipate @ 300mg/kg body weight with the second dose at 14 days after the first treatment. It was advised to change the litter as it was the source of infection. 14 days after the first treatment pooled droppings were examined for EPG. It was significantly reduced to 0-100. Mortality was also gradually reduced to 0.8 % after treatment.

4. Discussion

Infection with *A. numidae* was the cause of mortality and morbidity in this pearl variety guinea fowls. Absence of diagnostic lesions in organs other than intestines in necropsy, microscopic morphology of adult parasites, histopathology and response to piperazine treatment suggest this association. The identification of *A. numidae* was based only on the microscopic morphology of the adult parasite recovered during postmortem.

*A. numidae* infection is relatively host specific (Bush, 1990) but the entry of infection into this organized farm is unknown. Multiple stages of nematode parasite in the same host indicate continuous reinfection from the source of infection. Weight loss and emaciation in *A. numidae* infected birds which has been reported might be due to diarrhea and alteration in the feed conversion ratio.

Tissue stages in the development of *A. numidae* from the guinea fowl has been reported from Georgia as early as 1973 (Mabon and Reid, 1973). Larval stages of *A. numidae* were found in the mucosa and submucosa has also been reported by the above authors. The hyperplasia of goblet cells in the mucosa and submucosa has been reported in *A. galli* infection in chickens (Soulsby, 1986). But it has not been previously reported in guinea fowls with *A. numidae* infection. Lesions in the liver and presence of cluster of bacilli in the intestine observed on histopathology might be secondary to the damage caused by penetrating larvae. No significant bacterial growth from the routine heart blood swab culture confirms the above finding. No other lesions in the liver, lung and other internal organs of the host coincide with the report of Mabon and Reid (1973).

There is a report on response to piperazine treatment for *Ascaridia numidae* infection (Robbins et al., 2011). Treatment with Levamisole @ 30 mg/kg body weight (Avcioglu et al., 2008), Fenbendazole have been suggested (Robbins et al., 2011). But in our study birds did not respond to Albendazole treatment.

Guinea fowl rearing is an important alternate poultry farming practiced in Indian subcontinent to utilize the poorly cultivable lands and this practice generate employment opportunity and revenue to the rural youth besides catering to the protein needs of the rural farmers. This report on *A. numidae* infection is first of its kind in Indian guinea fowls. This paper emphasizes the economic importance of *A. numidae* infection in guinea fowls as it causes heavy morbidity and mortality.

Prophylactic measures such as periodical removal of litters, stacking of litters for several days to allow heating before placing them in pens, good ventilation, feed trough and drinking water hygiene along with treatment may be useful in effective control of *Ascaridia numidae* infection in guinea fowls.

5. Conclusion

This study reported a significant mortality rate of 3.5% in grower age group of guinea fowl due to *Ascaridia numidae* infection. The diagnosis was established through identification of eggs in feces, EPG of feces and numerous adult worms in the intestine. Histopathology revealed a severe damage
to mucosa and submucosa of intestine by second stage larvae. Treatment with piperazine and prophylactic measures significantly decreased the mortality rate.

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