



ORIGINAL ARTICLES

Studies on Interactive Effects of Ochratoxicosis and Coccidiosis on Mortality, Lesion Score and Fecal Oocysts Count in Broiler Chickens

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ABSTRACT

The study was conducted by experimental induction of ochratoxicosis and coccidiosis individually and in combination in 4 groups of broiler chickens each consisting of 50 birds. Ochratoxicosis was induced by feeding of ochratoxin A (OA) 1ppm in the diet and coccidiosis was induced by oral inoculation of 50,000 sporulated oocysts of *Eimeria tenella* culture on 21st day. There was mortality of 3, 4, 14 and 16 percent in groups I, II, III and IV respectively. Significant increase in lesion score and fecal oocysts count was also observed in combined group at 7 days of post infection, possibly due to additive and immunosuppressive effect of OA and *Eimeria* oocysts.

Key words: Coccidiosis, *Eimeria tenella*, Ochratoxicosis, Ochratoxin A

Introduction

Poultry industry in India has achieved a phenomenal progress during the last few decades. Genetic improvement, scientific managerial practices and disease prevention program have been contributed to this vertical growth. In intensive poultry farming, birds are exposed to several stress factors which enhance their susceptibility to various diseases that are already existing or emerging. The birds are also susceptible to various non infectious agents and among which mycotoxicosis is most common and a persistent problem. Many mycotoxins mainly ochratoxin A specifically affect the lymphoid organs resulting in immunosuppression (Singh *et al.* 1990).

Coccidiosis is one of the most common protozoan disease affecting poultry. *Eimeria tenella* is the most common and highly pathogenic in nature resulting in field out breaks causing great economic loss to the poultry farmers. The concurrent occurrence of ochratoxicosis and coccidiosis in broilers has been reported by Huff and Ruff (1982).

Materials and Methods

A total of 200, day old unsexed broiler chicks were procured from a commercial hatchery and reared under optimal managerial conditions. On seventh day the chicks were randomly divided into four groups of 50 chicks each. Chicks belongs to group I healthy control, group II ochratoxin control, group III coccidia control and group IV received both ochratoxin and coccidia oocysts. Ochratoxin A (OA) was produced on broken wheat using *Aspergillus ochraceus*, NRRL 3174 culture as suggested by Trenk *et al.* (1971) with minor modifications and Naturally infected birds with caecal coccidiosis was selected during the post mortem examination in the department of Veterinary Pathology, Veterinary College, Bangalore and the oocysts were isolated.

Groups II and IV supplemented with ochratoxin A throughout the study and group III and group IV infected with *Eimeria* oocysts on 21st day by oral route. Six birds from groups I, II, III and IV were sacrificed on 5th, 7th, 9th and 11th day of post infection to study different parameters.

Lesion scores were made based on the severity of intestinal changes at the time of necropsy (Johnson and Reid 1970) with the following scale.

Score	Lesion
0	No gross lesion
+1	Very few scattered petechiae on the caecal wall; No thickening of caecal wall; Normal caecal content present.
+2	Lesions more numerous with noticeable blood in the caecal contents; Caecal wall somewhat thickened; Normal caecal content present.
+3	Large amount of blood or caecal core present; caecal walls greatly thickened; Little, if any, faecal contents in the caecum.
+4	Caecal walls greatly distended with blood or large caecus core; faecal debris lacking or included in the core.
	Dead birds scored as +4.

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Oocysts excretions per gram of feces were counted from the infected birds on 7th, 9th and 11th day of post inoculation.

Results and Discussion

The broiler chicks fed with a diet containing one ppm of OA showed four per cent mortality. The present observation is in accordance with those of Gibson *et al.* (1989) and Kubena *et al.* (1994). In the present experiment, coccidia infected groups showed 14 per cent mortality. However, Gardiner *et al.* (1954) reported 10 percent mortality in coccidia infected group.

In the present experiment, increased mortality encountered in groups III and IV attributed to prior damage to caecal mucosa by OA and subsequent infection of coccidia resulting in acute haemorrhages in the caecum. (Muthusamy *et al.*, 2004). The group IV which has received both coccidia oocysts and OA showed a significant increase in oocysts output when compared to the group which received coccidia oocysts alone (group III) which was mainly due to the severity of coccidial infection in presence of immunosuppressive effects in birds due to OA. The present results are in agreement with Huff and Ruff (1982). The group IV which has received both coccidia oocysts and OA showed an increase in lesion score when compared to group which received only coccidia Oocysts (group III), which was mainly due to the severity of coccidial infection in presence of Immunosuppressive effect by OA. (Huff and Ruff. 1982).

Table 1: (mean±SE) oocysts count in broiler chickens infected with *Eimeria* oocysts and its combination with ochratoxin A

	7 th dpi	9 th dpi	11 th dpi
Group III	7.02±0.130 ^a	2.56±0.721 ^a	1.017±0.038 ^a
Group IV	9.25±0.125 ^b	4.258±0.968 ^b	1.859±0.56 ^a

The mean values bearing common superscript within column did not differ significantly

Table 2: (Mean±SE) lesion score in broiler chickens infected with *Eimeria* oocysts and its combination with ochratoxin A

	5 th	7 th dpi	9 th dpi
Group III	4±0.25 ^a	3.5.0±0.25 ^a	1.5±0.29 ^a
Group IV	4±0.25 ^a	4.±0.25 ^a	2.5±0.26 ^a

The mean values bearing common superscript within column did not differ significantly

The higher mortality, lesion score and fecal oocysts count in groups III and IV mainly due to the role of OA and *Eimeria* oocysts. The combined groups VI and VIII showed more severe lesions including greatly distended caeca with unclotted blood and mucosal tissue debris with severe haemorrhagic typhlitis from five upto 7dpi. Similar lesions in ochratoxicosis and coccidiosis were well documented by Huff and Ruff (1975) and Muthusamy (2004). Further it was opined that the increase in severity of lesions could be due to direct damage to the intestinal mucosa and immunosuppression by OA. Which also histologically evidenced by severe hemorrhage with numerous second generation schizonts, matured merozoites and developing oocysts in crypts and submucosa.

It was concluded that the expression of coccidia was higher with the ochratoxin compared to coccidia alone

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