Vet.Med.J.,Giza. Vol.42,No.3.(1994):25 - 29.

INFLUENCE OF DEXAMETHASONE ON IMMUNITY AGAINST EIMERIA TENELLA INFECTION IN CHICKENS

BY

SAFAA H. HEGAZI, SALWA F. AWADALLA, EMILY F. NAGUIB and BOTHINA A. BADAWY

Poultry Dept. Animal Health Research Institute, Bokki, Egypt.

Received : 28/9/1994

SUMMARY

The present investigation was undertaken to study the effect of dexamethasone (DEX) on immunity against *Eimeria tenella (E. tenella)* infection in chickens. Three groups of one day-old coccidia free chicks were used. Group 1 was kept as non-infected control. group 2 was infected with 10,000 sporulated cocysts of *E. tenella* per chick and is considered as infected group. The 3rd group received 10,000 Eimeria oocysts per chick and was injected intramuscular with 0.1 mg dexamethasone every other day and is considered as infected dexamethasone group. The experimental chickens were challenged with 15,000 *E. tenella* oocysts. The results indicated the following findings:

1- Weight gain percent of infected chickens and infected treated with DEX was significantly lower than control.

2- The total oocysts output of *E. tenella* was significantly higher in the group of infected chickens treated with DEX than control and infected groups. The lowest *Eimeria* oocysts count was noticed in the group of infected chickens.

3- There was a significant decrease in weights of spleen, bursa Fabricius and thymus of infected chickens and infected chickens treated with dexamethasone. The lowest weights of spleen, bursa and thymus was recorded in infected chckens treated with DEX.

4- Serum total proteins, sabumin, alpha, beta, and total globulins were significantly decreased in infected group and infected chickens treated with DEX in comparison with control, while there was a significant increase in gamma globulin i n infected group. The A/G ratio of infected chickens was decreased than infected chickens treated with dexamethasone and control groups.

INTRODUCTION

The genus *Eimeria* contains a number of obligate intracellular protozoan parasites with a complicated life-cycle involving both asexual and sexual stages of development. Coccidiosis is caused by *Emeria* infecting primarily the intestine of the susceptible host, thereby seriously impairing the growth and feed utilization of livestock and poultry (Rose and Kesketh, 1991).

Joyner and Davies (1960) reported that the intestinal and cecal hemorrhages are the major symtoms of acute infections with *E. tenella* and *E. necatrix* in chickens.

Awadalla et al. (1994) reported that weight gain percent of chickens infected with cryptosporidium and *E.tenella* was significantly lower than control.

In mammalian systems, glucocorticoids have been shown to exert an inhibitory effect on concanvalin A (Con A)- induced or phytohemagglutinin induced T lymphoproliferation (Sekellick and Marcus, 1986).

Gross et al. (1979) found that chickens treated with glucocorticosteroids showed decreased

weight gain, atrophy of the spleen, thymus and bursa of Fabricius and decreased antibody response to sheep erythrocytes.

Long and Rose (1970) and Rose (1970) reported that a prolonged treatment of chickens with dexamethasone (DEX) enhanced disease susceptibility to *Eimeria* spp., higher oocytst production, prolonged patent period and a decrease of intestinal lymphocyte number were noticed. Furthermore, turkey Eimeria which normally show a strict host specificity, developed in chickens treated with dexamethasone (Mc Loughlin, 1969).

Isobe and Lillehoj (1992) found that various effect of glucocorticosteroids on the avian immune syptem were examined in chicken treated intramuscularly with 0.1 to 2.5mg dexamethasone or prednisolone. Kinetic changes in body weight gain, percentages of lymphocyte subpopulations and T-cell functions were examined following treatment with dexamethasone or prednisolone every other day. Chickens treated with dexamethasone or prednisolone showed a decrease in body weight gain. The total number of splenic lymphocytes of chickens treated with the two drugs was significantly lower than control. Splenic T cells obtained from dexamethasone treated chickens showed a significant depression in concanavalin A-induced lymphoproliferation and interleukin 2 and gamma interferon production. The results characterize a variety of immunosuppressive effects of glucocorticoids on the avian immune system.

In view of the selective effects of glucocorticoid on a variety immunological events, dexamethasone serve an important role in dissecting various complex immunologic phenomens. To provide an insight into the cellular mechanisms involved in glucocorticosteroid-mediated enhancement of disease susceptibility to *Eimeria tenella* in chickens, we investigate the effect of dexamethasone on immunity against *E. tenella* infection in chickens.

MATERIAL AND METHODS

Thirty, on day-old coccidia-free chicks were used for this study. The chicks were kept under hygienic condition in cages. They were divided into three equal groups. Group 1 was kept as non-infected control. Groups 2 was orally infected with 10,000 sporulated oocysts of *E. tenella* per chick (Dr. Amal H. T. Abd El-Nasser, Det. of Poultry Disease, Fac. Vet. Med. Zagazig University, Benha branch) by a stomach tube to assure direct enoculation of the infective stage of the parasite into the crop and in considered as infected group.

The3 rd group recieved 10,000 sporutated oocysts per chick of *E. tenella* and was injected intramuscularly with 0.1 mg dexamethasone (SIGMA) per chick every other day (7 injections) and is considered as infected dexamethasone group. The chickens of all groups were challenged with 15,000 sporulated oocysts of *E. tenella* per chick at 14 days of age.

Feces were collected on days 5 through 10 after challenge in separated containers, weighed and mixed thoroughly with a mixer. Oocysts counts of *E. tenella* were made using saturated salt flotation technique (Long et al., 1976). All feces were processed within 48 hours after collection.

All chicks were weighed before experimental infection and at the end of the experiment. Overall weight gain during the experimental period was calculated.

Serum samples were collected into clean dry vials after 10 days postchallenge and kept in -20°C until use for determination of total proteins according to the method of Gornall et al., (1949) and protein electrophoretic pattern according to the method of Bicter (1964).

Killing of the experimental chickens was done at the end of the experiment. Spleen, bursa and thymus were separated and weighed.

Table (1): Effect of dexamethasone (DEX) on weight gains and oocysts output ($x10^5$) per gram of feces from chickens infected with *E. tenella*.

Group	Oocysts count ($x10^5$)	% Weight gained
Control	$0.53^b \pm 0.03$	$70.48^a \pm 4.48$
Infected	$0.28^a \pm 0.05$	$53.35^b \pm 4.82$
Infected treated DEX	$0.76^c \pm 0.04$	$23.33^c \pm 2.45$

Different letters of means in the same column show significant differences at P< 0.01.

Table (2): Effect of dexamethasone (DEX) and *E. Tenella* infection on spleen, bursa and thymus weights in chickens.

Group	Organ weight (g)		
	Spleen	Bursa	Thymus
Control	$0.15^a \pm 0.01$	$0.38^a \pm 0.03$	$0.56^a \pm 0.03$
Infected	$0.14^a \pm 0.01$	$0.21^b \pm 0.02$	$0.33^b \pm 0.03$
Infected treated DEX	$0.06^b \pm 0.01$	$0.09^c \pm 0.02$	$0.22^c \pm 0.02$

Different letters of means in the same column show significant differences at P< 0.01.

Table (3): Total and electrophoretic patterns of serum proteins in chickens infected with *E. tenella* and treated with dexamethasone (DEX).

Group	Total protein g%	albumin g%	Globulins g%				
			Alpha	Beta	Gamma	Total	A/G Ratio
Control	3.24 ± 0.10	1.47 ± 0.05	0.32 ± 0.04	0.43 ± 0.11	1.02 ± 0.01	1.77 ± 0.06	0.83
Infected	$2.33^* \pm 0.18$	$0.88^* \pm 0.05$	$0.14^* \pm 0.03$	$0.09^* \pm 0.01$	$1.22^* \pm 0.02$	$1.45^* \pm 0.08$	0.61
Infected treated DEX	$2.01^* \pm 0.05$	$0.91^* \pm 0.05$	$0.16^* \pm 0.02$	$0.10^* \pm 0.01$	$0.84^* \pm 0.06$	$1.10^* \pm 0.03$	0.83

Mean ± standard error.
*** Significantly from control at P<0.01.**

RESULTS

Tabel (1) showed that there was a significant decrease in body weight gain percent in infected chickens and infected treated with dexamethasone (DEX) group in comparison with non-infected control. The minimal weight gain percent recorded in infected chickens treated with dexamethasone (DEX). The infected chickens treated with DEX produced significantly higher of total *Eimeria* oocysts output than control and infected groups. The lowest *Eimeris* oocysts output was found in the group of infected chickens.

Table (2) reveals that there was a significant decrease in weights of spleen, bursa and thymus in infected group and infected chickens treated with DEX in comparison with control. There was no significant variation in weight of spleen of infected and control chickens. The lowest weight of spleen, bursa and thymus was noticed in infected chickens treated with DEX.

Table (3) indicatd that both groups of chickens infected with *E. tenella* and treated with DEX showed a significant decrease ($P< 0.01$) in serum total protein, albumin, alpha, beta, gamma and totla globuline in comparison with that of control, while there was a significant increase in gamma globulin in untreated infected chickens. The A/G ratio of infected chickens with *Eimeria* was decreased than control, while there was no alteration in infected chickens treated with DEX.

DISCUSSION

Concerning the effect of adminstration of dexamethasone (DEX) and *E. tenella* infection on body weight gain in chickens, the results of the present study showed clearly that the maximal gain in body weight was obtained in case of non infected negative control chickens. There was a significant retardation in weight gain percent in all infected chickens with *E. tenella* and infected treated with DEX group. These results could be attributed to the damage induced by the developmental stages of the parasites in the intestinal mucosa and submucosa and therefore impaired digestion, absorption and metabolic processes through the intestinal wall. These results are in agreement with those reported by Rose and Hesketh (1991), Isobe and Lillehoj (1992) and Awasalla et al., (1994).

The total *E. tenella* oocysts output was significantlly higher in the group of infectd chickens treated with DEX than that of control and infected groups. The lowest *Eimeria* oocysts output was noticed in the infected group. This might be due to the variety of immunosuppressive effects of dexamethasone on the avian immune system. These results were in agreement with that obtained by Long and Rose (1970) and Rose (1970), who found that a prolonged treatment of chickens with dexamethasone enhanced disease susceptibility to *Eimeria* spp., higher oocyst production, prolonged patent period and a decrease of intestinal lymphocyte number were noticed.

There was a significant decrease in weight of spleen, bursa Fibricius and thymus of infected chickens with *Eimeria* and infected chickens treated with DEX. The lowest weight of spleen, bursa and thymus was recorded in treated chickens with DEX. This could be attributed to the fact that coccidia infection and dexamethasone causes atrophy of spleen, thymus and bursa in chickens. These results were in agreement with those obtained by Gross et al. (1979).

Concernign the total and electrophoretic patterns of serum proteins in infected chickens with *E. tenella* and treated with dexamethasone. There was a significant decrease in total protein, albumin, alpha, beta, gamma and total globulins of infected chickens and the group of infected treated with DEX in comparison with control. This could be attributed that the glucocorticoid dexamethasone causes immunosuppressive effects on the avian immune system. These results were in agreement with those obtained by Isobe and Lillehoj (1992). There was a significant increase in gamma globulin in infected untreated chickens, the carrier of antibodies due to the *Eimeria* infection indicated a high sensitivity of chickens to produce antibodies for resisting such infection.

REFERENCES

Awadalla, S.; Badawy, B.; Naguib, E.; and Hagazi, S. (1994): Effect of concurrent infections with cryptosporidia and *Eimeria tenella* in chickens. Accepted in Vet. Med. Journal, Giza.

Bicter, B. W. (1964): Electrophoretic analysis of blood serum and plasma proteins of normal horses, Am. J. Vet. Res., 30: 2237-2241.

Gornall, A.; Bardawill, L. and David, G. (1949): Determination of total protein. J. Biol. Chem., 177: 751.

Gross, W. B.; Siegel, P. B. and Dubois, R. T. (1979): Some effects of feeding corticorterone to chickens. Poult. Sci. 516: 522.

Isobe, T. and Lillehoj, H. (1992): Effects of Corticorteroids on lymphocyte subpopulations and lymphokine secretion in chickens. Avian Diseases 36: 590-596.

Joyner, L. P. and Davies, S. F. M. (1960): Detection and assessment of sublethal infections of *Eimeria tenella* and Eimeria necatrix. Exp. Parasitol. 9: 243-249.

Long, P. L.; Joyner, L. P; Millard, B. J. and Norton, C. C. (1976): A guide to laboratory techniques used in the study and diagnosis of avian coccidiosis. Folia Veterinaria Latina 6: 201-217.

Long, P. L. and Rose, M. D. (1970): Extended schizogony of Eimeria mivati in dexamethasone treated chickens. Parasitology, 60: 147-155.

Mcloughlin, D. K. (1969): The influence of dexamethasone on attempts to transmit Eimeria meleagridis to chickens and *E. tenella* to turkeys. J. Protozool. 16: 145-148.

Rose, E. M. (1970): Immunity to coccidiosis : Effect of bethamethasone treatment of fowls of Eimeria mivati infection. Parasitology 60: 137-146.

Rose, M. E. and Hesketh, P. C. (1991): *Eimeria tenella:* Localization of the sporozoites in the caecum of the domestic fowl. Parasitology 102: 317-324.

Sekellick, M. J. and Marcus, P. L. (1986): Iduction of high titer chicken interferon. Methods Enzymol. 119: 115-125.