OnLine Journal of Biological Sciences 2 (10): 650-651, 2002
ISSN 1608-4127

Effect of *Ascaridia galli* Infestation on Electrolytes and Vitamins in Chickens

Haseeb Anwar and [1]Zia-ur-Rahman
Department of Veterinary Parasitology, [1]Department of Physiology and Pharmacology,
University of Agriculture, Faisalabad-38040, Pakistan

Abstract: Worm establishment in the intestine of chickens exposed to singly or multiple infection of *Ascaridia galli* has been used as the main criteria for judging the immune status to infections with nematodes. The change in the permeability of intestine may affect the availability of nutrients. One hundred one-day-old white leg horn chicks were reared for 28 days. Then two groups of 50 chicks each were made. Group I was kept uninfected (control) and group II was orally infested with 350 embryonated eggs of *Ascaridia galli* in gelatin capsule. After slaughtering the birds at 7,14, 21 and 40 days serum and liver samples were collected for the establishment of electrolytes and vitamin from each group. No significant different were observed in body weight and in the concentration of vitamin A and carotenoids of serum and liver at 7 and 14 days but theses values decrease significantly at day 21 and 40 in infected group. These changes attributed to the damage of intestinal wall and thus have decreased absorption of electrolytes and vitamins.

Key words: *Ascaridia galli*, chickens, electrolytes, vitamin

Introduction

Ascaridiosis is still a cause of economic losses in modern poultry (Permin and Ranvig, 2001). Worm establishment in intestine of chicken exposed to single or multiple infections of *Ascaridia galli* (*A. galli*) has been used as the main criterion for judging their immune status to infection with the nematode. Birds aged more than 3 months are resistant to the infection in comparison to birds of younger age groups. Even marked variations in susceptibility to infection, based on intestinal worm establishment, have been reported in birds of varying age groups (Subramanian and Sing, 1973; Pavlicek and Dykova, 1975). Dhar and Raina (1987) reported that smaller the infectivity dose, the higher is the worm populations and with an increase in the size of the infectivity dose, there is a corresponding fall in the establishment of worms in the intestine. Ikeme (1971a,b) demonstrated experimentally that infected fowl developed diarrhoea, hemoconcentration, intestinal lesion which are more severe in birds given poor diet. However, when the amount of protein in feed was increased, a significant variation in body weight between infected birds was demonstrated. Hiba and Geneidy (1965) studied that the functions and permeability of the intestinal wall were affected by the size of the *A. galli* infestation in five weeks old chickens.

The changes in the permeability of intestine may affect the availability of nutrients. Therefore, the present investigation was undertaken to evaluate the effect of *Ascaridia galli* infestation on the electrolyte and vitamin concentration of chicken.

Materials and Methods

One hundred, one-day old white leghorn chicks were reared in clean and sterilized cages under helminth free conditions, fed on a balanced ration at the Poultry House, Department of Physiology and Pharmacology, University of Agriculture, Faisalabad during the month of February-March. The chicks were fed on starter ration of 28 days and then replaced with finisher ration. Birds were divided into two groups of 50 chicks each. Group I was kept as non-infected thus served as control. Group II was orally infested with 350 embryonated eggs of *A. galli* in gelatin capsule and administered to each bird, followed by a wash with water.

Ten chickens, from each of the experimental and control groups were randomly selected and slaughtered at 7, 14, 21 and 40 days post infection (PI). Their serum and liver samples were collected for the establishment of electrolytes and vitamins. One ml of the serum was digested by sulphuric acid and perchloric acid (Rihcard, 1968). Digestion mixture was diluted to 25 ml of deionized water and preserved in plastic bottle till analysis at room temperature. Electrolyets (Na, K) were estimated using flame photometer while magnesium was estimated using atomic absorption spectrophotometer. Inorganic phosphorus was determined according to the method of Garher and Miller (1983). Serum and liver vitamin A and carotenoids were determined according to the method described by Cerr and Price (1926) with some modification (Varley *et al.*, 1976). The collected data was statistically analyzed using the student's t-test (Steel and Torrie, 1988).

Results and Discussion

No significant difference was observed in body weight at 7 and 14 days of infestation, however, the body weight decreased significantly at 21 and 40 days of *A. galli* infestation (Table 1). Such decrease in body weight might be due to destruction of the intestinal mucosa. Awadalla (1998) demonstrated glandular necrosis of the submucosa induced by the migration of larvae during the tissue phase and the presence of *A. galli* during the intensinal phase. Such damage causes disturbances and interference in the overall absorption, thus altering metabolic processes of nutrients through the intestinal wall. Teodorova and Gabrashanska (2002) suggested an optimal therapy containing a pure Cu basic salt and an organic Mn compound to correct minerals deficiencies and pathological symptoms and ensured lower mortality and higher gain body weight. Gauly *et al.* (2002) demonstrated that growth rate of the white hens was significantly higher in the control group as compared to chicks infected artificially with 250 embryonated *A. galli* eggs. Ramadan and Abou Zanda (1991) studied the pathogenicity of *A. galli* for young rossbroiler chickens fed different doses of infective eggs. They reported that biochemical analysis of muscle and liver tissue of the chicken showed a decreased in both glycogen and protine content when compared with those of normal tissue.

Table 1: Average body weight of chicken affected by *A. galli*

	Body weight (g)	
Post infection days	Control	Infected
7	85.6±2.0	86.0±1.5
14	168.0±1.0	165.0±1.3
21	680.0±4.0	674.0±3.0
40	1805.0±6.0	1609.0±8.6*

*Significantly different from control value of $P<0.05$

No significant difference was observed between the control and *A. galli* infested birds in the concentration of sodium, potassium, calcium, magnesium and inorganic phosphorous (Table 2). On day 21 and 40 PI, serum sodium, potassium and calcium concentration were significantly lowered and magnesium and inorganic

Corresponding Author: Haseeb Anwar, Department of Parasitology, University of Agriculture, Faisalabad-38040, Pakistan

Table 2: Effect of *A. galli* infection on the serum electrolytes of chicken

	Post infestation days							
	7		14		21		40	
Parameters	Control	Infected	Control	Infected	Control	Infected	Control	Infected
Sodium (mEq/L)	140.00±2.50	141.20±3.00	139.00±2.30	14.00±1.6	144.00±2.8	13.00±3.5*	142.00±2.1	131.00±3.8*
Potassium (mEq/L)	4.80±0.22	4.60±0.30	5.80±0.38	6.00±0.24	5.91±0.40	3.00±0.30*	5.60±0.27	3.14±0.21*
Clacium (mg %)	16.25±0.80	17.00±1.10	17.00±0.66	17.00±0.86	18.80±0.80	15.00±0.42*	19.50±0.46	15.00±0.50*
Magnesium	2.60±0.08	2.55±0.11	2.70±0.13	2.40±0.20	2.73±0.16	92.96±0.14	2.70±0.16	3.04±0.18
Phosphorous (mg %)	4.80±0.05	4.82±0.04	5.80±0.03	5.60±0.04	6.33±0.05	6.90±0.05	6.40±0.04	7.82±0.06

Table 3 : Effect of *Ascaridia galli* infestation on serum and liver vitamins of chicken

	Post infestation days							
	7		14		21		40	
Parameters	Control	Infected	Control	Infected	Control	Infected	Control	Infected
Serum Vit. A (IU/dl)	74.60±2.20	72.80±1.88	76.00±1.86	77.00±2.0	77.00±1.24	74.64±1.03*	76.60±0.96	73.15±1.26
Liver Vit. A (IU/dl)	232.00±4.11	231.80±3.61	230.00±4.16	229.00±3.27	231.00±2.80	219.00±1.68*	235.00±3.63	168.00±4.0
Serum carotenoids (IU/dl)	8.10±0.09	8.00±0.12	7.81±0.08	7.92±0.08	8.60±0.14	6.60±0.13*	8.80±0.16	6.50±0.16
Liver carotenoids IU/dl)	16.25±0.30	15.80±0.26	17.50±0.45	16.89±0.62	17.89±0.22	14.08±0.32	18.06±0.42	14.00±0.40

*Significant different from control value within the respective day of infestation day at P< 0.05

phosphorous were significantly higher during the intestinal phase of infestation as compared to control birds. The changes in electrolytes and trace elements are indicative of pathological catabolic processes that have been contributed due to the effect of *A. galli* on the middle part of small intenstine, where absorption of minerals occurred. Ramadan and Abou Zanda (1991) demonstrated that at autopsy, the small infestive showed external macroscpoic lesions of haemorrhage and congestion. In addition, intestinal obstruction with adult *A. galli* was also found in the infected birds. There was no significant variation in the concentration of vitamin A and carotenoids of serum and liver at 7 and 14 days between control and *A. galli* infested with chickens (Table 3). However, these values decreased significantly on day 21 and 40 in chickens infested with *A. galli*. These post infestation changes that also represent the intestinal phase of parasite are attributed to the damage of intestinal wall that might have decreased absorption of vitamin A as well as carotenoids by the infested birds.

From this study it can be concluded that chicken infested with *A. galli* have lost a significant body weight due to the lack of absorptions of nutrients, electrolytes and vitamins as a result of intestinal obstruction.

References

Awadalla, S.F.,1988. Studies on the effect of *Ascaridia galli* on the intestinal and blood parameters in chickens. Ph.D. thesis, Fac. Vet. Med., Cairo, University, Egypt.

Cerr, C.C. and E.A. Price, 1926. Colour reaction attributed to vitamin A. Biochem. J., 20: 497-502.

Dhar, D.N. and O.K. Raina, 1987. Establishment of *Ascaridia galli* in betamethasone-treated chickens. Vet. Parasitol., 25: 67-73.

Garher, C.C. and R.C. Miller, 1983. Preivisions of the 1963 semidine HCL standard method for inorganic phosphorus. Clin. Chem., 29: 184.

Gauly, M., C. Bauer, R. Preisinger and G. Erhart, 2002. Genetic differences of *Ascaridia galli* egg output in laying hens following a single does infection. Vet. Parasitol., 103: 99-107.

Hiba, M.H. and A.A. Geneidy, 1965. The effect of ascaridiasis in some trace elements and mineral level in serum of Egyptian chicken. Z. Parasitkde, 25: 399-404.

Ikeme, M.M., 1971a. Effects of different levels of nutrition and continuing dosing of poultry with *Ascaridia galli* eggs on the subsequent development of parasite populations. Parasitol., 63: 223-250.

Ikeme, M.M., 1971b. Weight changes in chicken placed on different levles of nutrition and varying degrees of repeated dosage with *Ascaridia galli* eggs. Parasitol., 63: 251-260.

Pavlicek, J. and I. Dykova, 1975. Experimental infection with *Ascaridia galli* in young chickens of different age. Acta Vet. Brno, 44: 223-233.

Permin, A. and H. Ranvig, 2001. Genetic resistance to *Ascaridia galli* infections in chickens. Vet. Parasitol., 102: 101-11.

Ramadan, H.H. and N.Y. Abou Zanda, 1991. Some pathological and biochemical studies on experimental ascaridiasis in chickens. Nahrung, 35:71-84.

Richards, L.A., 1968. Diagnosis and Improvement of Saline and Alkaline Soil. 1st Ed. Agri. Hand Book No. 60. IBH Publication Co. New Dehli, India

Steel, R.G.D. and J.H. Torrie, 1988. Principles and Procedures of Statistics: A Biometrical Approach. 2nd Ed. McGraw Hill Book Publishing Inc. New York, USA.

Subramanian, G. and K.S. Singh, 1973. Use of nuclear techniques in the control of *Ascaridia galli* in India. Investigations on immunization of one week old chicks with irradiated eggs. In: Isotopes and Radiation in Parasitology III. IAEA Vienna, pp: 67-71.

Teodorova, S.E. and M. Gabrashanska, 2002. Optimal treatment of *Ascaridia galli* infected chickens with salts of trace elements and a kinetic model for chicken growth. J. Helminthol., 76: 79-85.

Varley, H., A.H. Gowenlock and M. Bell, 1976. Practical Clinical Biochemistry 5th Edit. Williams Heinemann Medical Book Ltd. London, pp: 215-217.