Effects of *Ascaridia galli* infection and Cadmium Acetate Treatment on Tand B Lymphocytes in W.L.H. Chicks

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Abstract

Male White Leg Horn chicks were given infection of Ascaridia galli eggs dose (1000 eggs) and Cadmium acetate treatment (5 mg. /100 ml) was given. After 30 days, blood was collected for Tand B Lymphocytes study. Agalliinfection and toxicant induce immunological alteration in blood. It specifies that humoral and cellular immune responses are modulated by parasites and toxicants.

Keywords:-*W.L.H. chicks, ascariasis, A galli, T Lymphocytes, B Lymphocytes, Cadmium acetate.*

Introduction

In our country poultry production has remained as backward venture till 1960, but now it has emerged into an encouraging enterprise for rural folk, especially for landless labourers, small farmers and educated unemployed persons and also for big enterpreneurs maintaining birds on large scale.*Ascaridia galli.* is an intra intestinal worm found in chickens, turkeys, geese and number of wild birds. *Ascaridia galli* leads to highest degree of pathogenicity.

Cadmium exposure appears to inhibit development of antibody production. Cadmium is toxic to a number of organs such as kidney, liver, bone, blood, intestine, spleen and immune system (Friberg *et al* 1986). Large Part of immunological work on chickens is considered with T cell and B cell system, which is based on differences in thymic and bursal influences. Various mechanisms which effect immune responses are B Lymphocytes, T Lymphocytes, macrophages, antibodies, natural killer cells, serum factors and other cytokine factors (Chandra, 1982). Blackwell *et al (*2001) suggested that B cells and antibodies are required for resistance to gastro intestinal nematode. *Trichuris muris*.Thymus dependent cells are associated with cellular immunity (delayed hypersensitivity) and bursal dependent cells provide humoral immunity.

Materials and Methods

The following heads were included in experimental aspects.

1. Collection of parasites
2. Culturing of A. galli eggs.
3. Administration of dose — 1000 embryonated eggs of *A galli* and cadmium acetate (5mg. /100 ml)

The following experimental groups were categorized as

Group I Control male chicks.

Group II Chicks infected with 1000 embryonated eggs of *A galli*.

Group III Chicks treated with 5mg/ 100ml of cadmium acetate.

Group IV Chicks infected with 1000 embryonated eggs of *A galli* + treated with 5mg/100ml of cadmium acetate.

Collection of blood:-

Male W.L.H. Chicks were sacrificed after 30 days of infection and treatment. Blood was collected from heart with sterilized dry glass syringe by cardiac puncture. Blood was used for separation of T and B Lymphocytes.

T cells and B cells were identified. For counting, 10 ml aliquot was kept on counting slides. Cover slip was kept over it.

Counting slide was kept under light microscope and T & B cells were counted counting of T cells and B cells were repeated three times and mean was taken.

$$\text{Percentage of T cells} = \frac{\text{Number of Tcells}}{\text{Total number of Tcells and Bcells}} \times 100$$

$$\text{Percentage of B cells} = \frac{\text{Number of B cells}}{\text{Total number of T cells and B cells}} \times 100$$

Results:-

Analysis of T. Lymphocytes

Group I in control group, percentage of T Lymphocyte were counted to be 65.99 percent after 30 days.

Group II Percentage of T. Lymphocytes for were 68 percent after 30 days of infection.

Group III Percentage of T lymphocyte were counted to be 61.57 percent.

Group IV Percentage of T lymphocyte were 64.91 percent after 30 days of post Infection and Post Treatment. The percentage of T Lymphocytes were observed to be significantly ($P < 0.005$) suppressed in comparison to control group indicating suppression of cellular immunity.

Analysis of B Lymphocytes

Group I Percenage of B Lymphocytes was found to be 30.20 after 30 days.

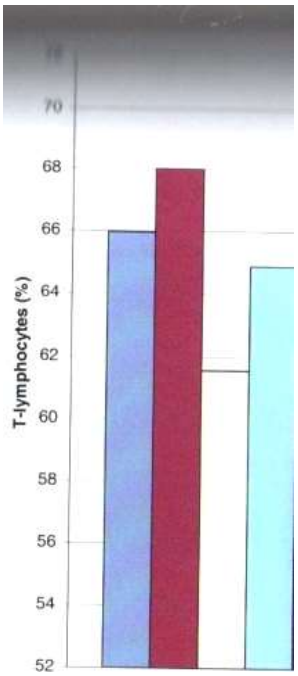
Group II Percentage was 30.65 after 30 days of infection.

Group III Percentage of B Lymphocyte was counted 34.81 percent after 30 days of Post treatment. A significant ($P<0.005$), rise was observed in comparison to control group depicting increased humoralimmunity.

Group IV Percentage was 34.86 after 30 days of post Infection and Post treatment. Percentage showed a significant ($P<0.005$) increase in comparison to control group, indicating elevated humoral immunity.

T Lymphocytes and B Lymphocytes, in Control, infected and treated W.L.H. chicks.

Groups	T lymphocytes %	B lymphocytes %
Group I	65.99 + 8.640	30.20 + 3.560
Group II	68.00 + 9.544	30.65 + 4.542
Group III	61.57 ± 8.629	34.81 ± 3.886
Group IV	64.91 ± 7.613	34.86 ± 2.650



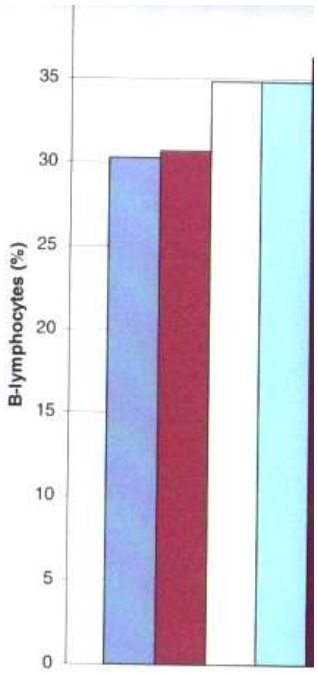
T - Lymphocytes (%) in control, infected and treated chicks.

Group I - Control Chicks

Group II - Chicks infected with 1000 embryonated eggs of *A galli*

Group III Chicks treated with 5 mg/100 ml of cadmium acetate

Group IV - Chicks infected with 1000 embryonated eggs of *A galli* and treated with 5 mg/100ml of cadmium acetate



B - Lymphocytes (%) in control, infected and treated chicks.

Group I - Control Chicks

Group II - Chicks infected with 1000 embryonated eggs of *A galli*

Group III Chicks treated with 5 mg/100 ml of cadmium acetate

Group IV - Chicks infected with 1000 embryonated eggs of *A galli* and treated with 5 mg/100ml of cadmium acetate

Discussion

Tand B Lymphocytes play important role in cellular and humoral immune responses. The number of B Lymphocytes cells were found highly increased in group infected with *A. galli* and in treated group. In present study, T cells were observed to be suppressed and B cells were augmented for production of antibodies against antigen. Thus, it indicates that both humoral and cellular immune responses are modulated by parasitic infection and environmental pollution. It provides maximum protection by modulating B and T cells in favor of host. On the basis of various experimental evidence, above studies provided that *A galli* infection and toxicant induce immunological alteration in blood. The knowledge regarding altered parameters would be useful in understanding immune system of the host.

Tew *et al* (1992) observed migrating of B cells from germinal center to bone marrow where they differentiate to long lived plasma cells and also contribute to prolonged elevation in antibody levels. A reduction in percentage of T cells and increase in B cells was observed by Canals *et al* (1997). He studied cytokine profile induced by a primary non-protective infection with *Ostealgia ostertagi* in cattle. This infection results in decreased level of IL-2 mRNA expression and increase in IL-4 and IL-10 transcription. Paciorkowski *et al* (2000) found that functional activity of B cells lasted longer and played a critical role in host protection against lymphatic filarial parasites. Lanyan et *al* (1996) performed observation on Kinetic changes of specific helper T cell subsets of mice after infection with *Trichinella spiralis*.

Rauw*et al* (2009) studied about humoral, cell mediated and mucosal immunity induced by Oculo-nasal vaccination of one

day old SPF and conventional layer chicks with two different live new castle disease vaccines.

Boroskova *et al* (1998) observed that free albendazole stimulated T cells and B cells activity to polyclonal activations.

Babu *et al* (1999) reported a more significant role of B cells in resistance to early phase of experimental murine filariasis. The elucidation of mechanism by which B cells mediate clearance of B. *malayi* infection was significant in developing vaccine molecule. Lev Kul *et al* (1999) observed functional activity of T cells in lambs after infection of *Ascaris suum*.

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