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Immunopathological Changes Induced By Sensitized Spleenic Cells in Spleen of W.L.H Chicks During Experimental Ascaridiasis

Zoology

KEYWORDS : WLH-White leg horn, PI-Post infection, PCI-Post challenge infection

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ABSTRACT

Newly hatched WLH chicks were infected by Ascaridia galli eggs with 1500 eggs and 3000 eggs subsequently immunized with spleenic cells. After 15 and 30 days of post infection spleen showed various pathological changes with high dose of infection. After 15 days of PI the T.S passing through the spleen revealed mild depletion of lymphocytes, distinct secondary module and parallel non- inflammatory edema in red pulp .The reticular tissues revealed prominent dowdy swelling. The red and white pulp area showed depletion of lymphocytes. Venous sinuses were highly dilated .After 30 days of PI the capsular wall was thickened with wavy outline. White pulp revealed dilated spleenic blood vessel and depletion of lymphocytes and capsular walls was ruptured at various places and white pulp showed inflammatory edema while in the immunized chicks the spleen not showed changes.

INTRODUCTION

Parasitic infections induce immune responses immunosuppressant and immunopathological changes. *A.galli* causes gastero-intestinal disease of poultry (freeborn 1923) Immunization have emerged strong second line of therapy. *A.galli* is an intestinal worm and chicken under three month of age are mostly susptible to it. Larval stage of *A.galli* causes anaemia, hyperproteinemia and several pathological disorders.

MATERIALS AND METHODS

Newly hatched WLH chicks were procured from Salim hatchery Meerut.Chick were housed in clean cages in animal house. All chicks were provided feed and water property twice a day. Donor chicks were sensitized by infecting them 1500 and 3000 eggs of *A.galli* .After 15 days of cell transfer chicks were given challenge infection of 1500 and 3000 infective eggs. After 15 and 30 days of PCI the spleen gland was processed for immunospathological examination.

The experimental chicks were divided into the following groups:-

Group I- Healthy+ chicks
Group II- Chicks infected with 1500 embryonated eggs of A.galli

Group III- Chicks infected with 1500 embryonated eggs of A.galli.+ challenged with spleenic cells.

Group IV- Chicks infected with 3000 embryonated eggs of A.galli.

Group V- Chicks infected with 3000. embryonated eggs of A.galli+challenged with spleenic cells.

Each group of chicks were regularly checked for activity, behavior,weight and other related condition. Chicks were anesthetized decapitated and autopsy was performed. Spleens were removed and placed in 10% neutral buffer formaline.Organn samples were processed by standard histopathological techniques.

Results and discussion

Group (I) –Structure of spleen (Healthy control) [Fig.1 and 2]

The transverse section of spleen from control group of chicks revealed following structures. The spleen has two regions: (1) Capsule and (2) Sub capsule. Capsule was made up of an outer and inner layer of collagen and elastic fibre. Subcapsule was broadly divided into two regions (a) Red pulp (b) White pulp

Red Pulp: Red pulp was observed to be a loose sponge tissue composed of ramifying cellular cords surrounded by venous sinuses. Venous sinuses were observed to be passage lined by flattened and elongated littoral cells. The tissue of red pulp area was basically composed of reticular cells and their fibres with a number of scattered large lymphocytes and macrophages.

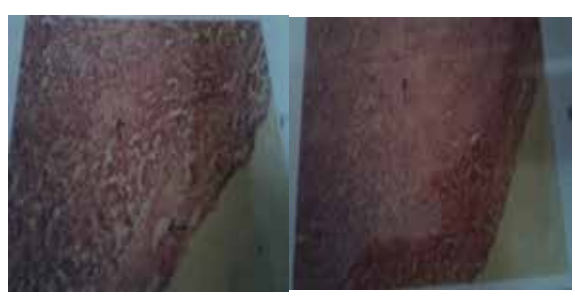

Fig. I and 2-T.S passing through the spleen of group I at 15th and 30th day showing Red pulp area , White pulp area and Capsular area.

White Pulp: White pulp observed to be diffused area and predominantly surrounded the spleen arteries. The white pulp area surrounded by red pulp tissue and having small and medium sized lymphocytes.

Group (II)- Infected with 1500 embryonated eggs of *A.galli*
15th day of PI,[Fig.3]

Here evident changes were observed in capsular wall that was sinusoidal congestion due to huge patches of red blood cells and few lymphocytes were focally infiltered in capsular wall, the red pulp area also became widely detached from capsular wall.

Fig 3-T.S passing through the spleen of group 2 at 15th day of Pi showing their sinusoidal congestion due to huge patches of red blood cells and red pulp details from capsular wall.

30th day of PI (Fig.4)

At some places the capsular wall became quite thick and ruptured .Cloudy swelling was observed in the white pulp area.The artery was very much dilated and rest part of white and red pulp area have abundant number of lymphocyte cells.

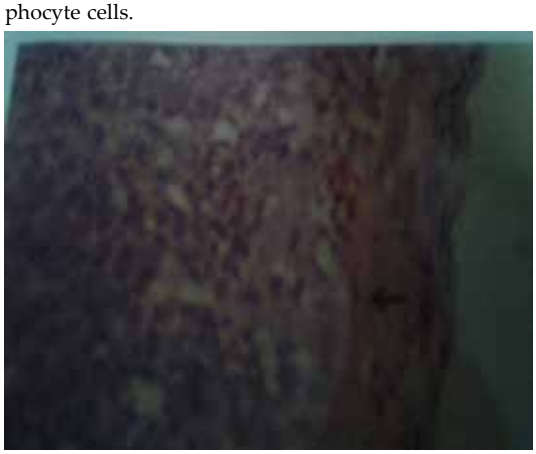

Fig 4-T.S Passing through the spleen of group 2 at 30th showing red pulp area and thick capsular wall

Group (III)- Immunized with sensitized spleenic cells and challenged with 1,500 embryonated eggs

15th day of PCi (Fig.5)

There was found to be thin capsular wall. Non-inflammatory edema was also observed in red pulp while white pulp observed to be normal in structure.

Fig.5-T.S passing through the spleen of group 3 at 15th day of PCI showing thin capsular wall and non-inflammatory edema

30th day of PCi (Fig.6)

After 30th day of PCI , the thick walled artery was found in white pulp area while red pulp area revealed a depletion of lymphocytes and congested sinuses at certain places.

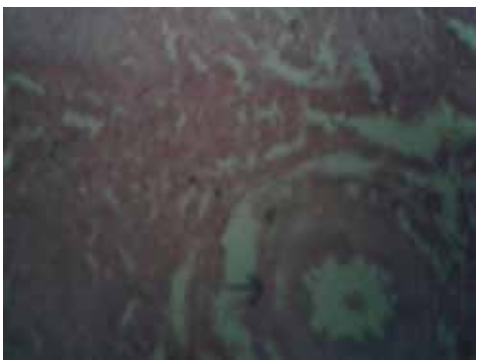

Fig. 6- T.S Passing through the spleen of group 3 at 30th day of PCi showing thick walled artery and depletion of lymphocytes cells in red pulp area.

Group (IV) Infected with 3000 eggs of a.galli

15thday of Pi (Fig. 7)

Well marked non – inflammatory edema was found in outer layer of capsular wall. Red pulp tissue was observed to be loosely packed spongy tissues, beneath the capsular wall , the depletion of lymphocytes were observed . Small arteries were also found in white pulp area.

Fig. 7 T.S passing through the spleen of group 4 at 15th day of Pi showing non- inflammatory edema in capsular wall, depletion of lymphocytes in red pulp area.

30th day or Pi (Fig. 8)

The capsular wall was found to be thickened and it revealed well marked inflammatory edema in outer and inner membrane. Hyperplasia of sheathed arteries were also observed in white pulp area .The red pulp and the white pulp have abundant number of lymphocytes but dilated venous sinuses was observed at some places of red pulp area.

Fig-8-T.S Passing through the spleen of group 4 at 30th day of Pi showing thick capsular wall inflammatory edema.

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Group(V) Immunized with sensitized spleenic cells and challenged with 3000 embryonated eggs
15th day of PCi (Fig. 9)

No significant structural changes were observed elastocollagenous capsule. Red pulp tissue possessed irregularly passages of venous sinuses while white pulp tissue was found to be diffused network of tissue. There was found numerous spleenic arteries and number of small sized scattered lymphocytes.

30th day of PCi (Fig. 10)

Capsular wall was found to be normal or regular in shape. There was no significant changes were observed in this portion. Here evident changes were found in red pulp area, the total depletion of

Fig.10-T.S passing through the spleen of group 5 at 30th day of PCi showing depletion of lymphocytes.

The present investigation have revealed that the experimental *A.galli* infection marked immunopathological changes in spleen of WLH chicks due to high dose of *A.galli*. The high dose of infected chicks revealed marked immunopathological changes marked hyperplasia of follicles were observed during investigation due to formation of secondary lymphoid nodule. The appearance of secondary lymphoid nodule were spleen associated with an infected but were absent in control chicks. Depletion of lymphoid cells were observed in spleen of broiler chicks(Stoeval at.2000;Tanigueri ,et at.,1977).Wedderburn (1974) observed hypertrophy of spleen. The changes in Jird spleens during the course of infection with B.pahangi and B.patei has been thoroughly examined by Vincent and Ash (1978). This organ exhibits hyperplasia and hypernemia in the host with trichinosis (Weatherly 1983 ;Singh and Rao,1987) . There is little specificity to such changes in lymphoid organs (Frizzera, 1986 ; Rideout et at., 1992).

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