Pakistan Journal of Nematology (2019) 37(1):103-105 ISSN 0255-7576 (Print)
ISSN 2313-1942 (Online)
www.pjn.com.pk
http://dx.doi.org/10.18681/pjn.v37.i01.p103-105

Larval migration of *Ascaridia galli* causes traumatic and toxic effects in chicken liver

S. Feroza[1], A. G. Arijo[1†], F. M. Bilqees[2] and M. S. Phulan[1]

[1]*Department of Veterinary Parasitology, Sindh Agriculture University, Tandojam, Pakistan*
[2]** Department of Zoology, University of Karachi, Karachi-75270, Pakistan*

[†]Corresponding author: abdullaharijo@sau.edu.pk

Abstract

Studies were conducted to assess the effects of infection on histological changes in liver of chicken, experimentally induced with *Ascaridia galli* round worm. The postmortem examinations of infected chicken revealed histo-pathological lesions of infected liver that showed traumatic and toxic effects produced by the migrating *A. galli* larvae. Except on the top, central veins were not obvious. Inflammatory tracts produced by migrating larvae were prominent. Large vein with portal sluggish consisted of fibrinous material and inflammatory cells, disarrangement of hepatici and shrinkage of hepatocytes were found to have resulted into dilation of sinusoids.

Keywords: *Ascaridia galli*, histopathology, chicken, nematode

The commercial poultry farming in Pakistan was started in 1960s. Due to its application, today, it has become a profitable industry and has acquired a very significant place in overall economy of Pakistan. *A. galli* has been reported as a cause of economic loss of millions of kilos of meat and tens of thousands of eggs in poultry (Peirce & Bevan, 1973). The parasite lives in the central portion of the small intestine of domestic fowl and other birds (Mark & Bisgaard, 2001). The most important pre-disposing factors are young age, coccidiosis and feeds deficient in vitamin A and protein (Gordon & Jordan, 1982). These parasites cause reduction in growth rate, weight loss and damage to the intestinal mucosa leading to blood loss and secondary infection (Gauly *et al*., 2002). Objective of this study was to assess the effects of infection on histological changes in liver of chicken, experimentally induced with *Ascaridia galli* round worm.

Materials and Methods

Collection of samples: In order to see the effect of infection on histo-pathological changes in chicken, 20 commercial chicken of 2 weeks age were reared and divided into 2 groups (a) Experimental birds (b) Control birds. Both groups were provided similar feeding, housing and related management. High positive *A. galli* eggs infection was induced in experimental chicken of group (a) and left for further 2 weeks for infection to establish. After 2 weeks, confirmation of infection was done through coproscopy. The samples collected were brought to the laboratory, Department of Veterinary Parasitology, Faculty of Animal Husbandry and Veterinary Sciences, Sindh Agriculture University, Tandojam. The infected tissues were processed for histo-pathological studies at Liaqat University of Medical and Health Sciences, Jamshoro, Sindh Pakistan.

Published by Pakistan Society of Nematologists
Received: 07 Feb, 2018
Accepted: 20 May, 2018

Histo-pathological examination: Morbid tissues from the liver were fixed in 10 % formalin, and further processed with ascending grades of ethyl-alcohol to prepare the paraffin blocks. Sections were cut at 5-6 microns thickness and slides were stained with Hematoxylin and Eosin (Elizabeth & Fredric, 2001).

Results

Histo-pathological lesions and poorly defined lobulations in case of traumatic and toxic effects were seen by the migrating larval migratory tracts. The liver demonstrated disarray of lobulations. Inflammatory tracts produced by migrating larvae were prominent. These migratory tracts had started from a vein which indicated haematogenous migration of larvae. One section of larvae have shown a large vein with portal slugging consists of fibrinous material and several migratory tracts starting from the vein, stained darkly representing accumulation of inflammatory cells, which were noted with atrophy of hepatocytes and shrinkage of hepatocytes that resulting into dilation of sinusoids (Fig. 1. B, D).

Other photograph (Fig. 1 C) shows part of a large vein partly obliterated. Larval migratory tracts starting from the vein and extending to liver parenchyma may also be seen. At the periphery of the vein on the right, accumulation of degenerated liver cells can be seen; lobulation is poorly demarcated with ill-defined central veins. Two larval migratory tracts lined by inflammatory cells, disarrangement of hepatocytes were found with prominent sinusoids in the vein. Giant cells resembling the condition Giant Cell Cirrhosis was clear. Moreover, prominent spaces around the group of hepatocytes resulting into dilated sinusoids were observed. Two larval migration tracts with numerous inflammatory cells are obvious, and macrophages and lymphocytes being most prominent. The limiting membranes of the veins have been seen broken and bound directly by hepatocytes, as compared to normal (Fig.1 A).

Discussion

Liver is the metabolic factory, detoxification unit and storage organ in vertebrates. Our findings indicate some gross and histopathological changes in liver of chicken caused by *A. galli*. The histopathological changes (Fig. 1B & D) in this study show traumatic and toxic effects produced by the migrating larvae. The liver showed disarray of lobulation except on the top central veins where they were not obvious. However, inflammatory tracts produced by migrating larvae were prominent. These migratory tracts started from a vein; which indicates hematogenous migration of larvae and dis-arrangement of hepatocytes have been seen with prominent sinusoids, degenerated liver cells in lobulation of the liver. These conditions may be associated with entry of *Ascaris* larvae into hepatic portal veins and finally settling into hepatic parenchyma. More or less similar findings were noted by Abrha & Yohanns (2014) who reported liver tissues demonstrating diffused hemorrhage, mild to moderate degeneration, sinusoidal hemorrhage, congestion, focal necrosis, cellular swelling and vacillation in hepatocytes, and Kupffer cell hyperplasia. These findings were in agreement with those of Soulsby (1982) and Urquhart *et al*., (1996) who also noted same condition. Interestingly, they also noted heavy infection with larvae often produces intense inflammation, with edema and dis-arrangement of hepatocytes and degeneration of the liver cells in the inflammatory reaction.

It is a matter of concern as to how the parasite of GIT affects the liver. Nice convincing answer (as also proved from present study) came from Pritchard & Brown (2001) who reported that occasionally larvae may migrate to liver.

References

Abrha, B. & Yohanns, T. (2014). Clinical, gross and histopathological study on common local chicken diseases in Enderta District, South East Tigray. *European Journal of*

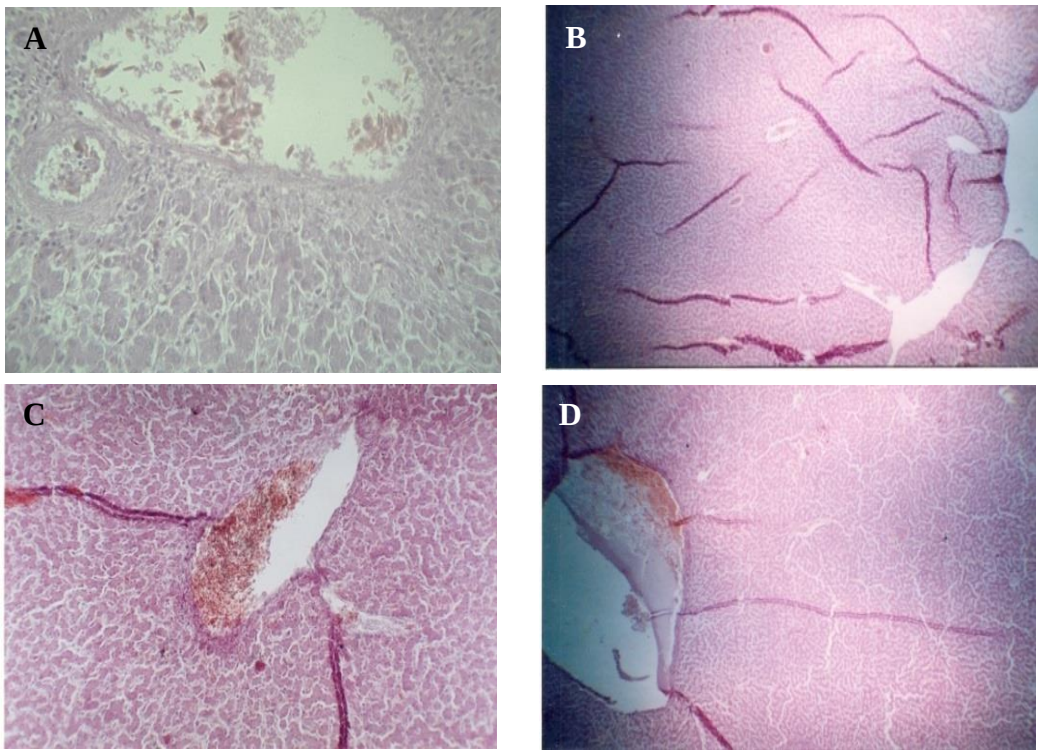

Fig.1 (A-D). A. Normal histology of liver; B. Microphotograph of liver section showing larval migratory tracts and poorly defined lobulations; C. Two migratory tracts starting from the vein, stained darkly representing accumulation of inflammatory cells, and the atrophy of hepatocytes; D. Microphotograph of liver section showing disary of lobulations. Except on the top central veins are not obvious. Inflammatory tracts produced by migrating larvae are prominent. These migratory tracts have started from a vein which indicates haematogenous migration of larvae. One section of larva can be seen in the vein. Partly obliteration of the vein by slugging is also obvious (Haematoxylin-Eosin; 10x).

Biological Sciences, 6, 95-103. DOI: 10.5829/idosi.ejbs.2014.6.04.85238

Elizabeth, A. & Fredric, L. F. (2001). Comparative veterinary histology with clinical correlates. CRC Press, London, 269pp.

Gauly, M., Baur, C., Preisinger, R. and Erhart, G. (2002). Genetic differences of *Ascaridia galli* egg output in laying hens following a single dose infection. *Veterinary Parasitology*, 7, 103. 99-107.

Gordon, R. F. & Jordan, F. T. W. (1982). *Poultry Backyard* (2nd Ed) Bailliere, London.

Mark, C. A. P. P. & Bisgaard, N. M. (2001). Investigation of the parasitic nematode *Ascaridia galli* (Shrank, 1788) as a potential vector for *Salmonella enteric* dissemination in poultry. *Journal of Parasitology Research*, 87, 317- 325.

Peirce, M. A. & Bevan, B. J. (1973). *Ascaridia galli* (Schronk, 1988). in psittacine birds. *Veterinary Records*, 92, 261.

Pritchard, D. I. & Brown, A. (2001). Is *Necator americanus* approaching a mutualistic symbiotic relationship with humans? *Trends Parasitology*, 17, 169-172. Doi: 10.1016/S1471-4922(01)01941-9

Soulsby, E. J. L. (1982). *Helminths, Arthropods and Protozoa of Domesticated Animals*. 7th Ed. East Sussex: Ballie´re Tindall, London, UK.

Urquhart, G. M., Armour, J., Duncan, J. L., Dunn, A. M. & Jennings, F. W. (1996). *Veterinary Parasitology*, 2nd Ed. Blackwell Science Ltd, Oxford. 307 pp.