

# Histopathological Changes in Liver of Albino Rat Induced by Experimental Infection of *A. lumbricoides*

Dr. Savita Rani

D.N. College, Meerut, India

**Abstract:** *Ascaris lumbricoides* is a gastrointestinal parasite that infects the digestive tracts of humans, causing Ascariasis, and is estimated to infect approximately 1.4 billion persons on a global basis. During the following investigations various immunopathological changes were observed in low and high doses of infection. Changes were evident in hepatic cells, sinusoids, central and portal vein. These were cloudy swelling, fatty cyst formation, kupffer cell hyperplasia and inflammation. In the present studies kupffer cell hyperplasia, focal collections of lymphocytes were observed in high dose of sixty days of post-infection.

**Keywords:** *Ascaris lumbricoides*, Albino rat, Hyperplasia, Inflammation

## 1. Introduction

*Ascaris lumbricoides* infection represents a major public health problem in poor and developing countries and has constituted a universal burden which does not only depend on regional ecological conditions but also on local standards of social and economic development of the people (Ukbaï et al, 2003). Ascariasis, an intestinal worm infection, is caused by the parasite *Ascaris lumbricoides* and a report by the World Health Organization (WHO) on soil-transmitted helminths suggests that over one billion people are affected by Ascariasis. This disease is prevalent in developing countries and in places of poor sanitation and unhygienic conditions. Even though anthelmintic drugs are available for the treatment of Ascariasis, it is considered as a neglected tropical disease.

## 2. Material and Method

The experiments were conducted on infection-free laboratory-maintained albino rats. The adult parasites were obtained from the intestine of freshly slaughtered sheep and kept in normal saline for egg laying. Sodium bicarbonate culture fluid was used for culturing *A. lumbricoides* eggs up to the embryonated stage. The experimental protocol comprises of a control group (6 rats), a second treated group administered a low dose of (500 embryonated eggs) of *A. lumbricoides* (6 rats) and a third group administered a high dose of (1000 embryonated eggs) of *A. lumbricoides* (6 rats). Two rats from each group were sacrificed after 20, 40, 60 days of post-infection. The tissues (liver) were further processed for microtomy and microphotographs.

## 3. Observation

Following changes were observed due to infection of *A. lumbricoides* in liver.

Changes in the liver of albino rats due to infection with a low dose (500 embryonated eggs) of *A. lumbricoides*

### After 20 days of post-infection

Hepatocytes showed a minimal degree of cloudy swelling. Sinusoids appeared normal and no Kupffer cell hyperplasia was seen. No specific alteration in the portal tract.

### After 40 days of post infection

Hepatocytes revealed degenerative changes, fatty cyst formation. The portal tract revealed fibrosis; inflammatory cells, mainly lymphocytes and macrophages.

### After 60 days of post infection

Hepatocytes characterized by feathery appearance of cytoplasm. Nuclear changes like cyanosis, karyolysis were observed. Sinusoids with Kupffer cell hyperplasia observed. Prominent fibrosis characterized by oblong nuclei seen around the portal tract.

### After 60 days of post-infection

Hepatocytes characterized by feathery appearance of cytoplasm. Nuclear changes like pyknosis, karyolysis and karyorrhexis were observed (fig 3). Sinusoids with Kupffer cell hyperplasia observed. Prominent fibrosis characterized by oblong nuclei seen around the portal tract (fig 4).

### Changes in the Liver of Albino Rats Due to Infection with High Dose (1000 EMBRYONATED EGGS) OF *T.OVIS*

### After 20 days of post-infection

Mild degree of perlobular fibrosis was recorded in hepatocytes. Dilatation of the central vein and sinusoids (fig 5).

### After 40 days of post-infection

Few lymphocytes were scattered diffusely in the liver lobule. Thick fibrous bands with prominent vessels and bile ductules were evident in the portal area (fig 6).

### After 60 days of post-infection.

Central vein and sinusoids appeared to be degenerative, necrotic changes were discerned. Kupffer cells seen in fair number. Focal collection of lymphocytes in the portal tract (fig 7).

#### 4. Discussion

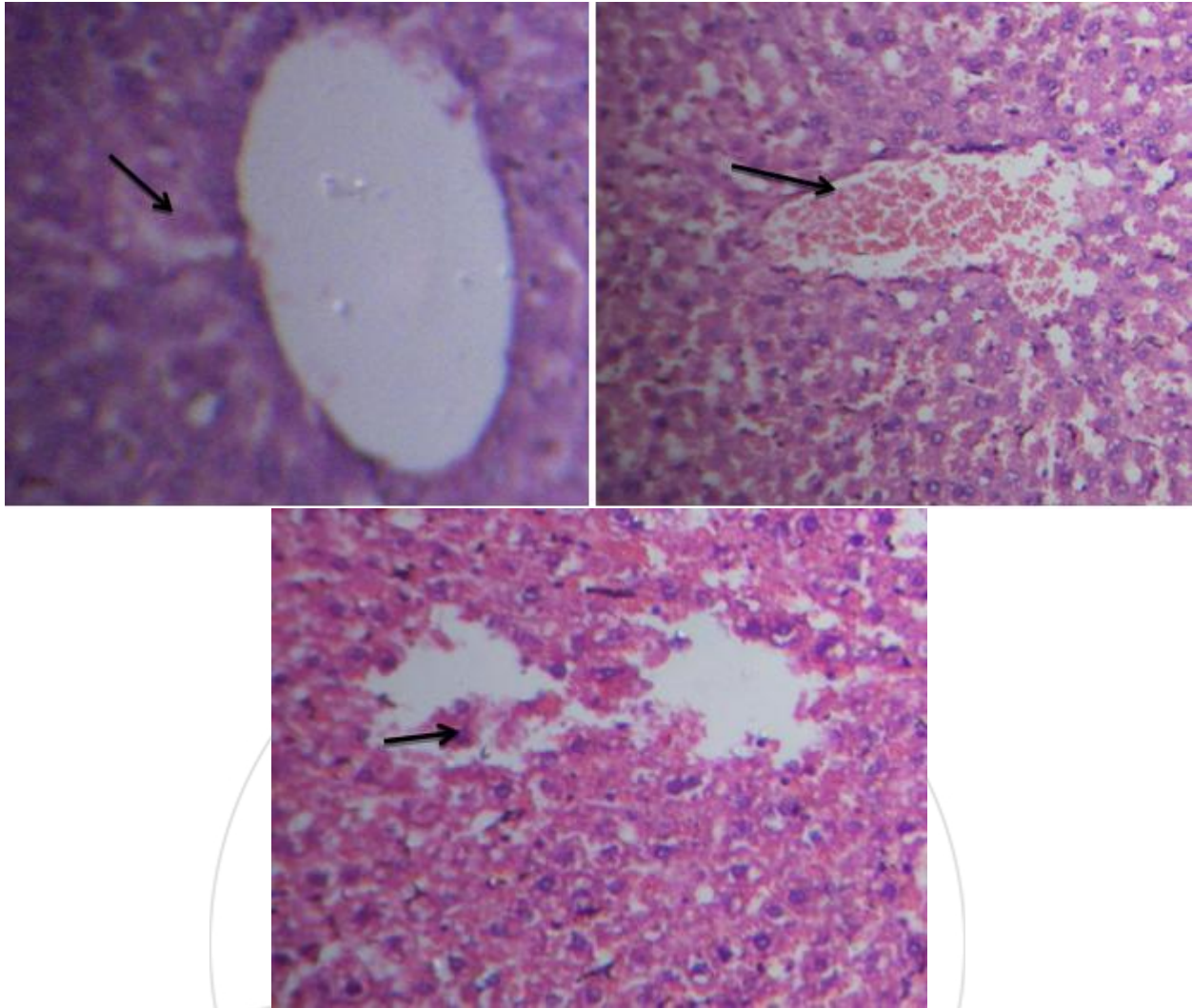
During the following investigations various immunopathological changes were observed in low and high doses of infection. Changes were evident in hepatic cells, sinusoids, central and portal vein. These were cloudy swelling, fatty cyst formation, kupffer cell hyperplasia and inflammation. Changes in hepatocytes have been reported by Mossoud (1981) in the sheep naturally infected with *Dicrocoelium dendriticum*. Fatty cyst formation in liver may be due to the interference in the fat metabolism of the liver. Dilation of central vein may be due to the normal tissue response towards internal parasite. Due to low and high dose of infection the liver of albino rats revealed cloudy swelling which is due to the toxins and antigen liberated by the parasite, an important pathological alteration in hepatocytes. In the present studies kupffer cell hyperplasia, focal collections of lymphocytes were observed in high dose of sixty days of post-infection. Pathomorphological changes were similarly observed in the liver of lambs after long term *Ascaris suum* infection (Krupicer et al 1999). Portal area revealed fibrosis, dilation and congestion sinusoids, degenerative changes in the nucleus. These changes were due to deficient blood supply, inadequate nutrition and oxygenation. Fibrosis in portal tract was observed by Radha et al (2003). Changes in the liver parenchyma was also studied by Perez et al (1999). Present author is of the opinion that various immunopathological alterations may be due to endotoxins liberated by the parasites. The stimulation of liver collagen synthesis and promotion of collagenolysis in schistosomiasis are thought to be indirect result of immunological host responses to schistosome egg antigen. A linkage between the parasite infection and these processes that take place between the parasite and the host's collagen metabolic pathways are responsible for above pathological changes. Anderson (2001) studied on ascaris that the dangers of using single locus markers in parasite epidemiology. Crompton (2001) and Bethony et al., (2006) worked on *Ascaris* and ascariasis. And studied on soil-transmitted helminth infections: *ascariasis*, *trichuriasis* and hookworm. Parvatham and Veerakumari, (2013) observed on Drug target prediction using elementary mode analysis in *Ascaris lumbricoides* energy Metabolism. Biotechnology and bioprocess engineering

#### 5. Acknowledgement

Authors are thankful to the Head Department of Zoology, D.N. College, Meerut for providing all the laboratory facilities.

#### References

- [1] Crompton, D.W.T. (2001): *Ascaris* and ascariasis. *Advances in Parasitology*, vol, 48, pp-285-375.
- [2] Jelliffe, D.B (1953): *Ascaris lumbricoides* and malnutrition, in tropical
- [3] Children. *Document? Jcimdicinecographica tropical.5* :314-320.
- [4] Bethony, J., Brooker, S., Albonico, M., Geiger, S.T., Loukas, A., Diemert, D., and Hotez, P.J. (2006): soil-transmitted helminth infections: ascariasis, trichuriasis and hookworm. Vol, 367, pp-6-12.
- [5] Krupicer, I; Ondrejka, R; Svicky, E; Vasilkova, Z; Dovroznakova, E; Dubinsky, P; Moncol, D.J. (1999): Clinical and pathomorphological changes in the organism of lambs after long term *Ascaris suum* infection. *Klinicke a patomorfbrogicke z meny v organizrnejhniai pod thodobominfikovani.vajickami Ascaris suum Solvensky Veterinarsky Casopis* (1999) 24 (2) 93-97
- [6] Massoud, J (1981): Histopathology of liver in Iranian sheep naturally infected with *Dicrocoelium dendriticum*. *Annals of Tropical Medicine and Parasitol.* 75(3): 293-298.
- [7] Perez, J; Martin de las Mulas, J; Carrasco, L; Gutierrez, P.N; Martinez-Cruz, M.S; Martincz-Moreno, A (1999): Pathological and Immunohistochemical study of the liver and hepatic lymph nodes in goats infected with one or more doses of *Fasciola hepatica*. *J. Comp. Patho.* Vol.120, 199-200. :
- [8] Radha, G; Ilurikrishan, T.J; Anna, T (2003): Pathology of experimental coccidiosis in lambs. *Indian Journal of Animal Sciences.* 73(5): 532-533
- [9] Smith, J.D (1908): Cited from "*Animal Parasitology*". Cambridge University Press.
- [10] Parvatham, k, and Veerakumari, L. (2013): Drug target prediction using elementary mode analysis in *Ascaris lumbricoides* energy Metabolism. *Biotechnology and bioprocess engineering* 18: 491-500.
- [11] Anderson, Tim, J.C. (2001): the dangers of using single locus markers in parasite epidemiology: *Ascaris* as a case study. Vol, 17, pp-183-188.
- [12] Tripathy, K; Kuque, E; Bolanos, O; Lotero, H and Mayoral, L.G (1972): *Clinical nutrition*, 25; 1276-1281.
- [13] World Health Organization (1967): Control of ascariasis, WHO. Technical Report no 314.



**Figure 1** show dilation & central vein, fig2, show, Necrosis.Mid cloudy swelling. Focal collection of Lymphocytes, hemorrhage in central vein. Fig 3, show inflammatory changes in hepatic cell sinusoids, Kupffer cell hyperplasia

