

HISTOPATHOLOGICAL ALTERATION IN THE LIVER OF MARINE EDIBLE FISH *LUTJANUS ARGENTIMACULATUS* (FORSK, 1775) DUE TO ACANTHOCEPHALAN PARASITE

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ABSTRACT

The present study was made to describe the cellular changes in the liver of edible fish *Lutjanus argentimaculatus* (Forsk., 1775) of Karachi coast, infected with acanthocephalan parasites belonging to genus *Serrasentis* sp. For the pathological observation of infected liver, slides were made by routine microtomical procedure and studied under microscope. The architectural changes of tissues due to acanthocephala are being described by the selected photographs. Focal necrosis, fibrinoid homogenous masses, atrophy, cirrhosis as well as vascular degeneration were noted. Proboscis penetrated in the liver due to which hepatocytes become atrophied and necrosis extended up to maximum surface of liver.

Keywords: *Lutjanus argentimaculatus*, *Serrasentis* sp., Acanthocephalan, Liver, Histopathology, Karachi coast.

INTRODUCTION

In the most diverse group of vertebrates, fish are important source of proteins and other essential nutrients throughout the world. This group is very rich in nourishment and consumed either in cooked, salted, smoked or preserved form. By-products of fish are also used for different purposes and are of great economic value. Such as, fish oil, fish meal, fish glue, isinglass, fertilizers etc. Occurrence of disease due to the parasitic infection in fish constitutes the most important problem and challenge worldwide. They are infected by viruses, bacteria, fungi and protozoans. They are also parasitized by various groups of helminths like monogeneans, digeneans, cestodes, nematodes and acanthocephalan (Khalid and Polling, 1997). All these parasites produce varying degree of pathogenicity (Paperna, 1996). Fish usually infected by these parasites show different diseases and alteration in their normal physiology (Kabata, 1985). Slow growth rate, abnormal activity, weakness, discoloration of skin, open sore etc. are the common signs of parasitic infection. The infected fish also lose their nutritional value. Most of these parasites infect humans by eating raw or insufficiently processed fish.

A number of reports are available on pathological changes of different organs of fish due to acanthocephalan infection (Hine and Kennedy, 1974; Bilqees and Fatima, 1992a,b; Bullock, 1963; Dezfuli, 1991; Dezfuli *et al.*, 2002a,b, 2008, 2009; Mc Donough and Gleason, 1981; de Buron and Nickol, 1994; George and Nadakal, 1981; Schmidt *et al.*, 1974; Sanil *et al.*, 2010; Sakthivel *et al.*, 2014).

Horny headed worms are usually recorded from the intestine of fresh and marine water fish. Very few reports found on the pathological changes due to helminths in various organs of fish *Lutjanus argentimaculatus*. Khatoon *et al.* (1999) and Rizwana *et al.* (2006, 2007) reported the histopathological changes in the stomach and intestinal tissue due to nematodes and trematodes, while Rizwana *et al.* (2011) observed architectural changes in the spleen of mangrove red snapper due to *Anisakis* larvae. Only two reports are available on the seasonal variation of helminth parasites in *L. argentimaculatus* by Rizwana *et al.* (1999), while another work on seasonal variation and histopathology of various organs of *Lutjanus argentimaculatus* was reported by Ghaffar (2007). Pathological manifestation of the intestine of mangrove red snapper by acanthocephala was reported by Sanil *et al.* (2010). No report is available on the pathogenicity of liver due to acanthocephalan parasite in the fish *Lutjanus argentimaculatus*.

MATERIALS AND METHODS

Mangrove red snapper, *Lutjanus argentimaculatus* (Forsk., 1775) collected from fisheries and various local markets of Karachi. These were brought to the laboratory of parasitology, Department of Zoology, University of Karachi for detailed study. Fish were carefully sized and examined for the helminth parasites. During the investigation six fishes out of twenty eight were severely infected by acanthocephalan parasites. Liver of infected

fish were severely damaged by the embedding of acanthocephalan proboscis and their mechanical movement. Entangled acanthocephala collected and processed by routine method. Mounted parasites were identified as *Serrasentis* sp. Infected sections of liver penetrated by worm proboscis were fixed in Buffer formalin for 24 hours then processed by routine method for microtomical studies. Tissue sections of 6-8 microns were cut and stretched on slides then deparaffinized and hydrated sections tissues were stained by H & E. After dehydration slides were mounted by Canada balsam and studied under microscope. Selected photographs of infected tissues were taken by photomicrographic camera.

OBSERVATIONS

Sections of the infected liver were showing exudates accumulation. The other connective tissue capsule was damaged and dislocated at several places. Pathological studies indicated severe destruction and necrosis of liver parenchyma. Atrophy, inflammation, hepatic architecture disintegration and fibrosis were the common findings. Severe caseation and focal necrosis were obvious in underlying tissues. Deterioration with immense involvement of macrophages and eosinophils were also noted (Figs. 1, 3, 4 and 5). Complete deformation of liver architecture with scattered destruction of hepatocytes with fibroid homogenous masses in the portal area was also prominent (Figs. 1, 3, 4, 5, 6 and 8). Ulcerative type lesions were obvious in critically affected areas (Figs. 5, 7 and 8). Acanthocephalan proboscis was prominent in the liver tissue (Figs. 1-3). Due to the proboscis perpetration, hepatocytes become atrophied (Fig. 2). The tissues surrounding the section of proboscis were shown to have large number of macrophages (Figs. 1-3). Penetrated hooklet of acanthocephalan proboscis was also observed (Figs. 7 and 8). The surrounding area of penetrated hooklet was severely damaged. Infiltrated macrophages, lymphocytes and other inflammatory cells in the surrounding necrotic area were prominent (Fig. 7 and 8).

Severe cell damages were observed in all sections. Morphological features of hepatocytes and hepatic cord were unidentifiable (Figs. 3-6). Necrosis around the portal areas in some sections were accompanied with fibrosis (Figs. 9-11).

Sections showed that the arteries lost their definite muscular arrangement as well as the normal layers of the wall become indistinct, consisting of homogeneous material with deterioration of fibrous layer (Fig. 8 and 9). Besides several eosinophilic cells were also obvious in the infected tissues.

DISCUSSION

Parasitic infection in fish cause heavy mortality in both marine and freshwater fish. Acanthocephala are usually found in the intestine of vertebrates but during the present study, these parasites were recovered from the liver. It is difficult to suggest whether they migrate through making tunnels from intestine into the liver or from intestine to visceral mesenteries on to the surface of liver. The present observations indicate the total destruction of hepatocytes and portal tract due to the proboscis penetration.

Taraschewski (2000) observed the degree of damage and intensity of host reaction due to acanthocephalan parasites which depends on the depth of parasitic penetration and further reported that acanthocephala produce severe damage to the liver and pancreas. Complete degenerative changes in the gut wall, liver and pancreas of fish were also observed by Akinsanya (2007). Acute inflammation in the peripheral and chronic inflammation in the deeper tissue was prominent. Dezfuli *et al.* (2009) also reported the inflammatory response in the fish tissue due to acanthocephalan parasites. At various sites of proboscis infestation, mostly eosinophils, lymphocytes and macrophages were present. Similar findings were presented by Dezfuli *et al.* (2015) in the liver of fish *Perca fluviatilis* infected by tapeworms. Santore *et al.* (2013) reported fibrosis associated with necrosis in antarctic fishes while the same changes were observed in the infected liver sections during present study. Necrosis, granulomas and ulcerative lesions were obvious. Another important finding was severely dilated sinusoid. Wanless (1994) suggested that dilation of sinusoid occur due to the atrophy of the hepatocytes, portal vein obstruction and disruption in the sinusoidal reticulin fibers.

Fish which are under stress and weaker are more susceptible and provide an ideal place for parasites to survive and flourish. The parasites not only affect the morphology of the infected organ but also interfere with nutrition and metabolism. Both these conditions result in disease and finally death of the fish. Parasites change the food preference of host fish which results in delayed growth and stunting (Palaq *et al.*, 2017).

The present observations showed severe architectural deterioration in the hepatocytes and tissues. The changes resulted into focal and general necrosis, ulceration as well as vascular degeneration. Same findings were also reported in different fishes infected with acanthocephalan parasites (Bullock, 1963; Mc Donough and Gleason, 1981; Dezfuli, 1991; Dezfuli *et al.*, 2002a, b, 2008; Irshadullah and Mustafa, 2012). Our observations, therefore, suggest a general impact on liver of fish by acanthocephala.

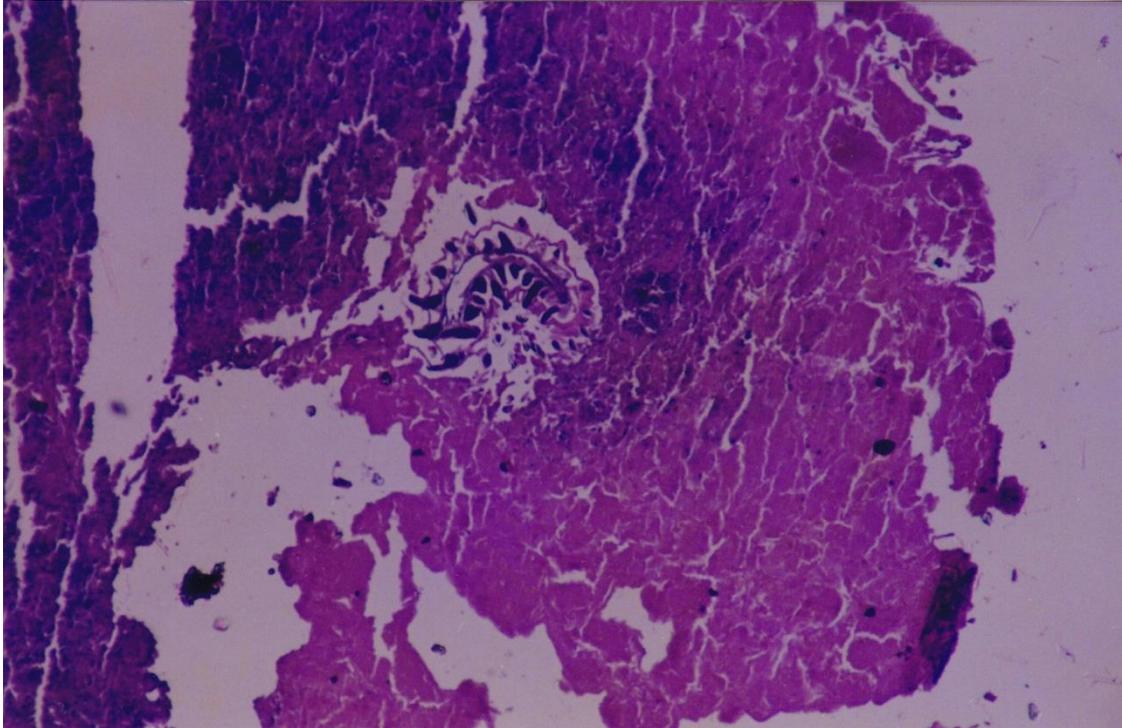


Fig. 1. Photomicrograph of portion of section of liver showing penetrated proboscis and severe host reaction (x 20).

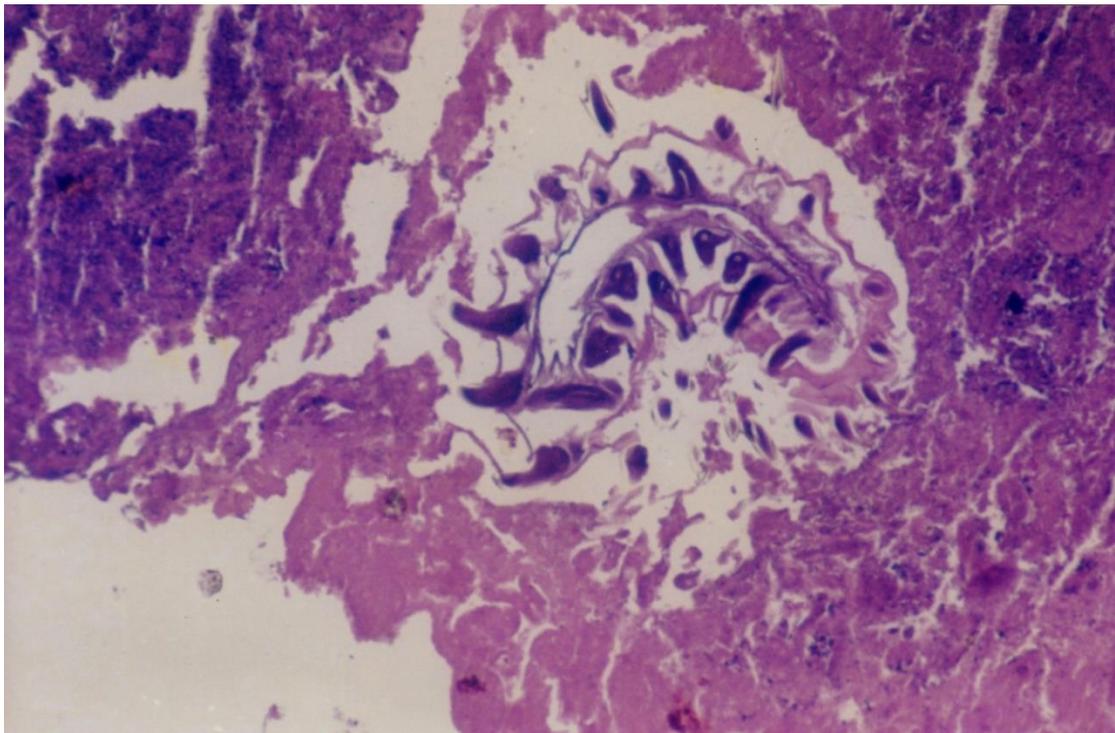


Fig. 2. Section of proboscis obvious penetrated in liver parenchyma, hepatocyte became atrophied (x 50).

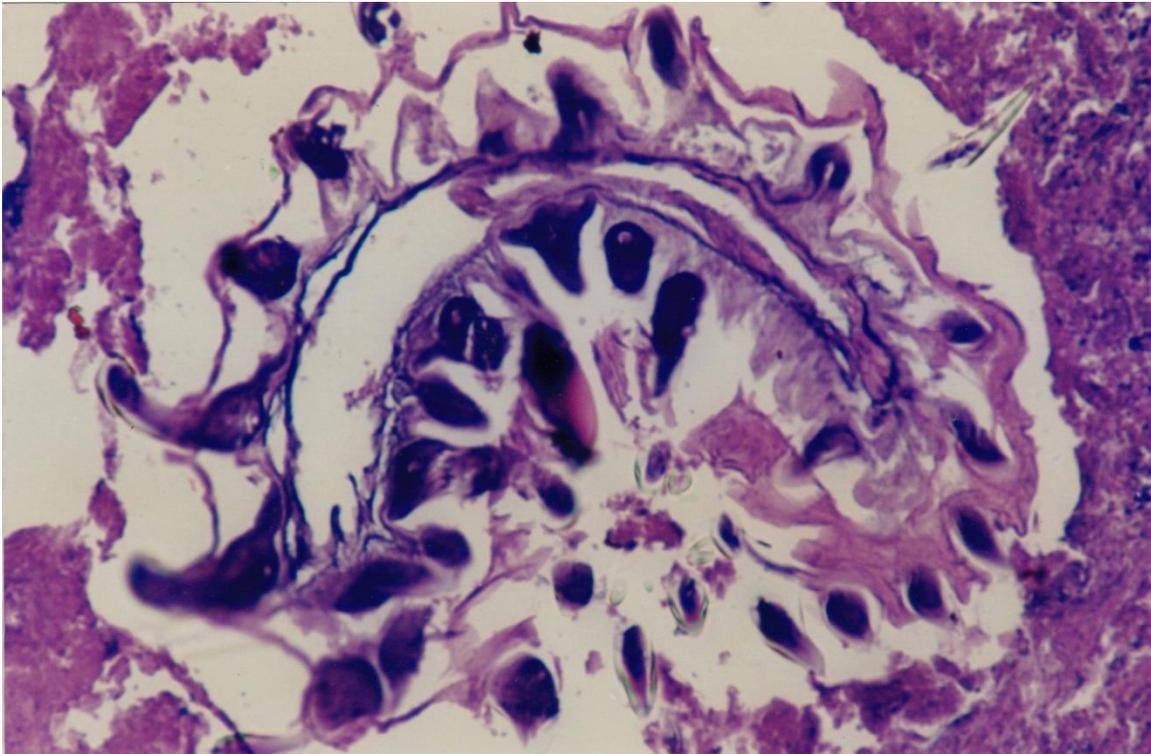


Fig. 3. Section of acanthocephalan proboscis as in Fig. 2 at higher magnification shows penetrated hooks (x 100).

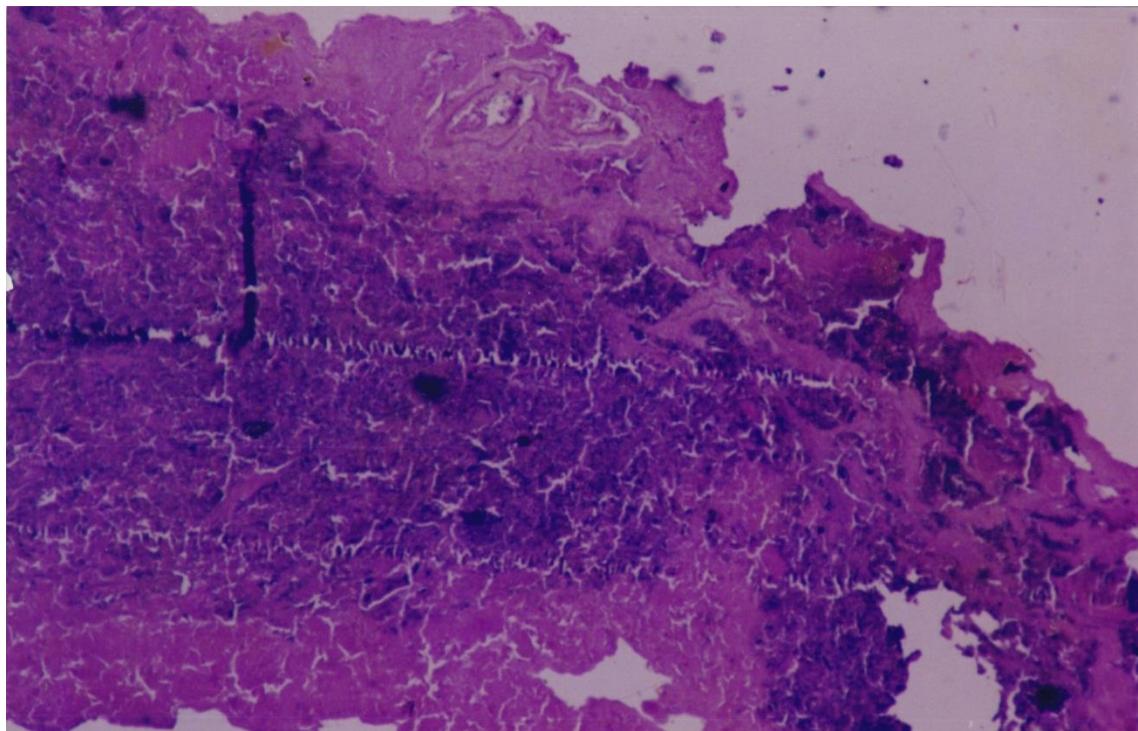


Fig. 4. Section of portion of liver shows atrophied hepatocytes and necrosis extends upto maximum surface of the liver (x 20).

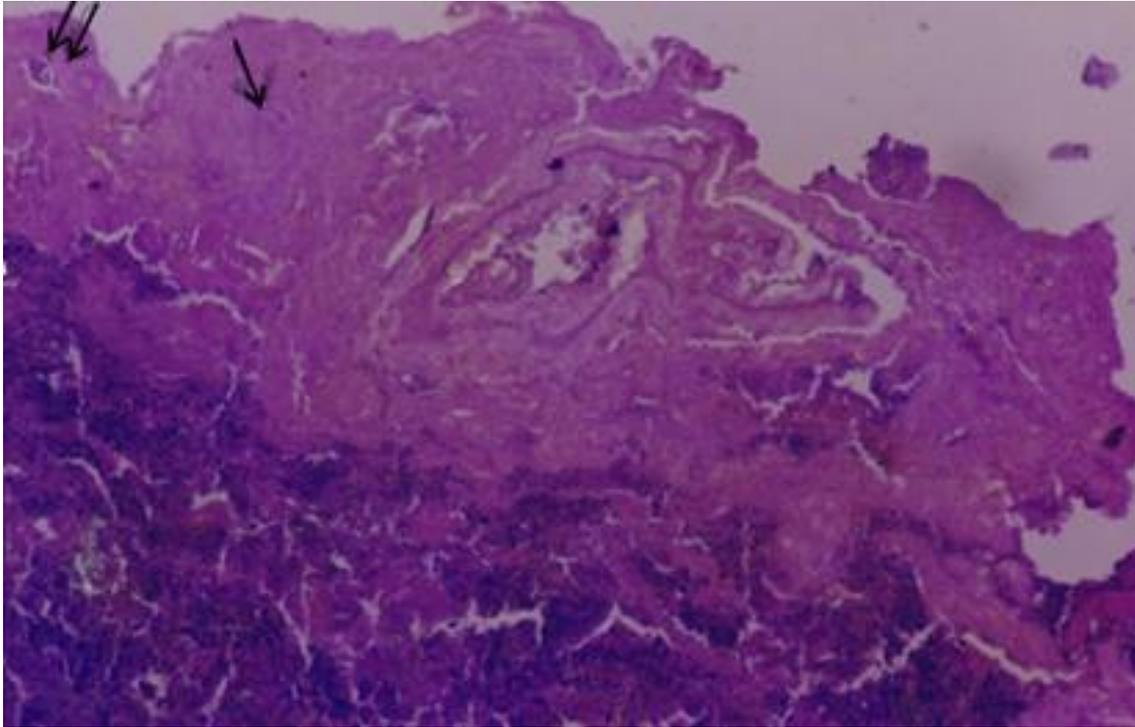


Fig. 5. Section of portion of liver shows infiltration of inflammatory cells and destroyed sinusoid due to acanthocephalan infection. Caseous necrosis is also prominent (arrow) at the site of penetrated proboscis hook (double arrow) (x 50).

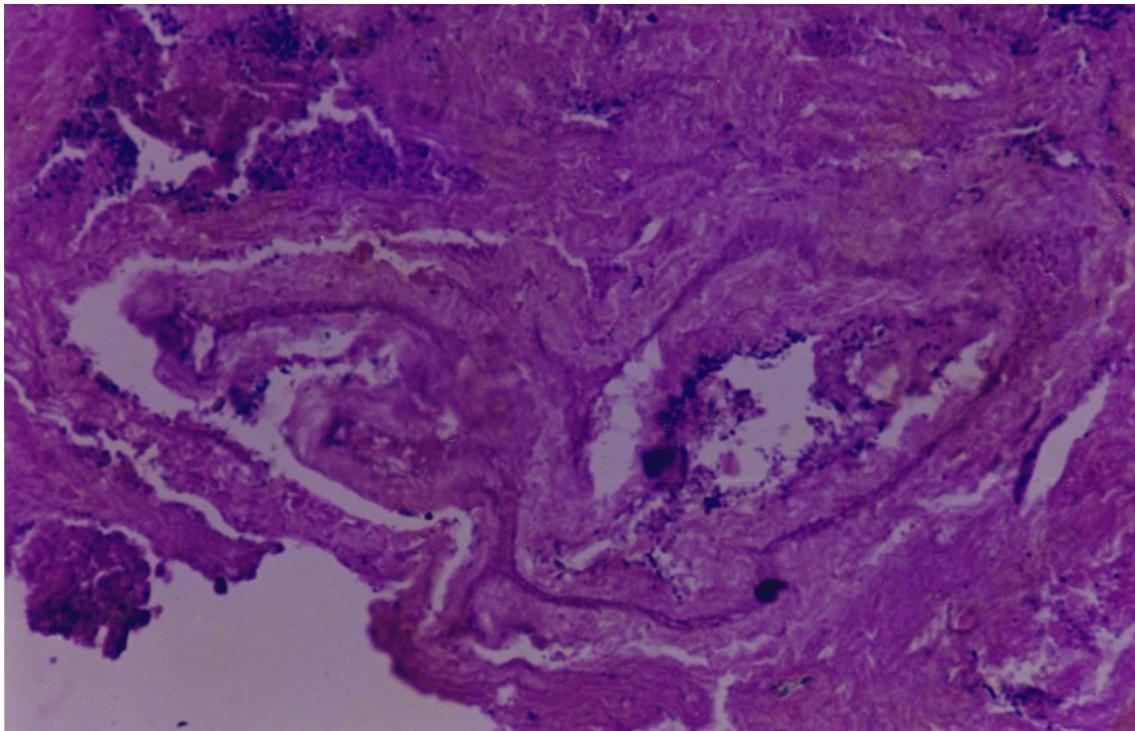


Fig. 6. Distended and necrotic sinusoid due to acanthocephalan infection. Whole surrounding area appeared necrotic and atrophied with the diminished of architectural morphology (x 100).

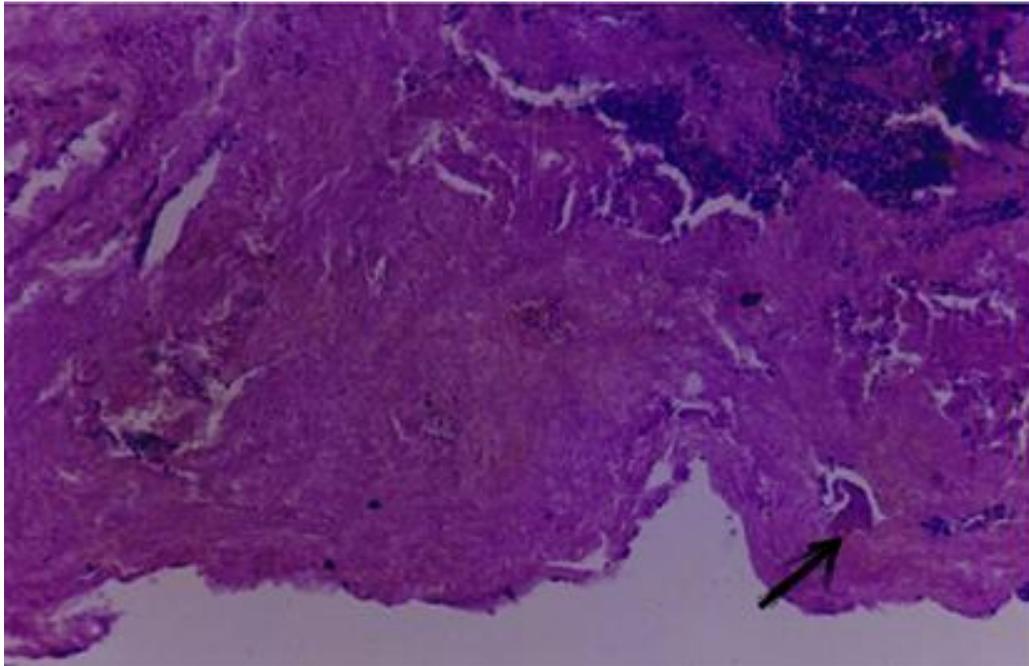


Fig. 7. Portion of section of liver shows penetrated hooklet (arrow), due to host-parasite response, the whole liver section appeared abnormal (x 100).

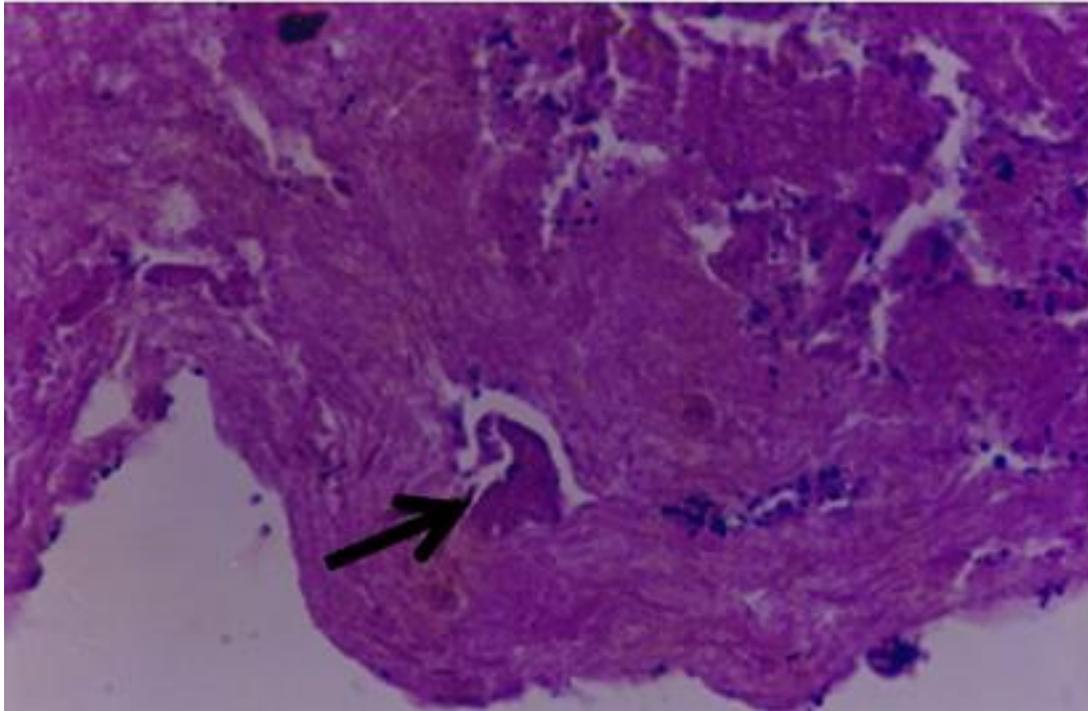


Fig. 8. Portion of liver as in Fig. 7 at higher magnification shows hooklet (arrow), infiltrated macrophages, lymphocyte and other inflammatory cells in the surrounding necrotic area (x 200).

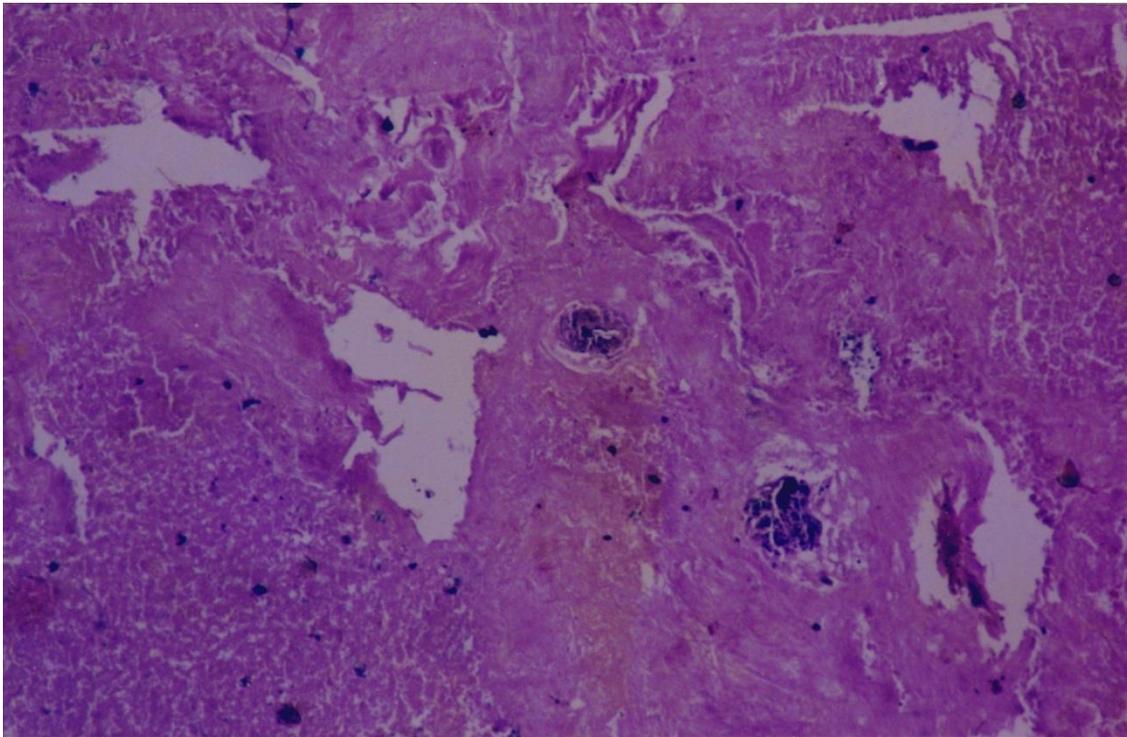


Fig. 9. Portion of section of liver shows caseous necrosis, granulomas and ulcerative lesions (x 20).

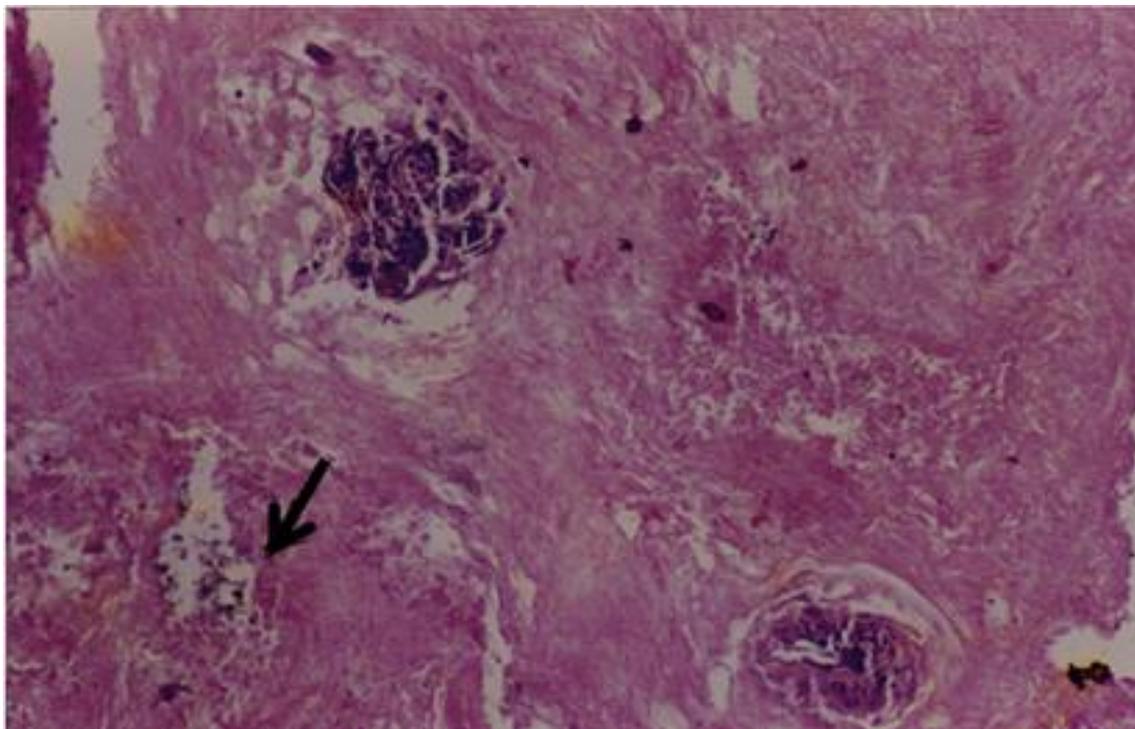


Fig. 10. Portion of liver as in Fig. 9 at higher magnification shows granulomas, caseous necrosis and necrotic foci (arrow) (x 50).

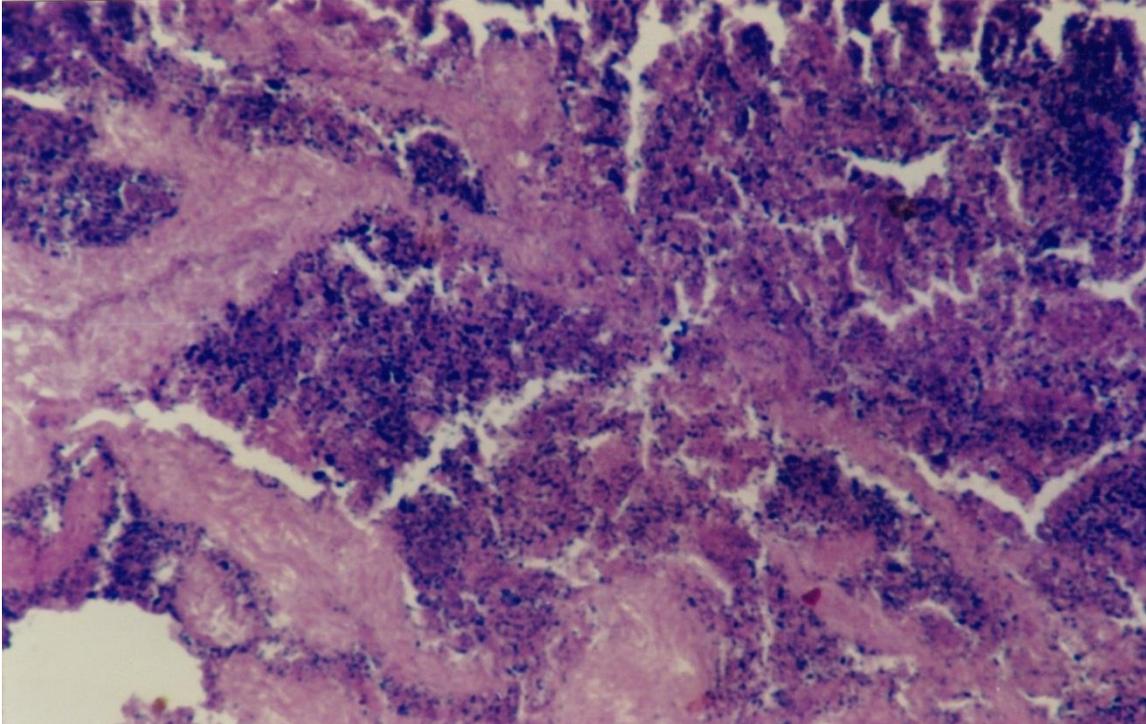


Fig. 11. Portion of liver shows extensive necrosis along with deviation of normal hepatocyte into homogenous masses (x 100).

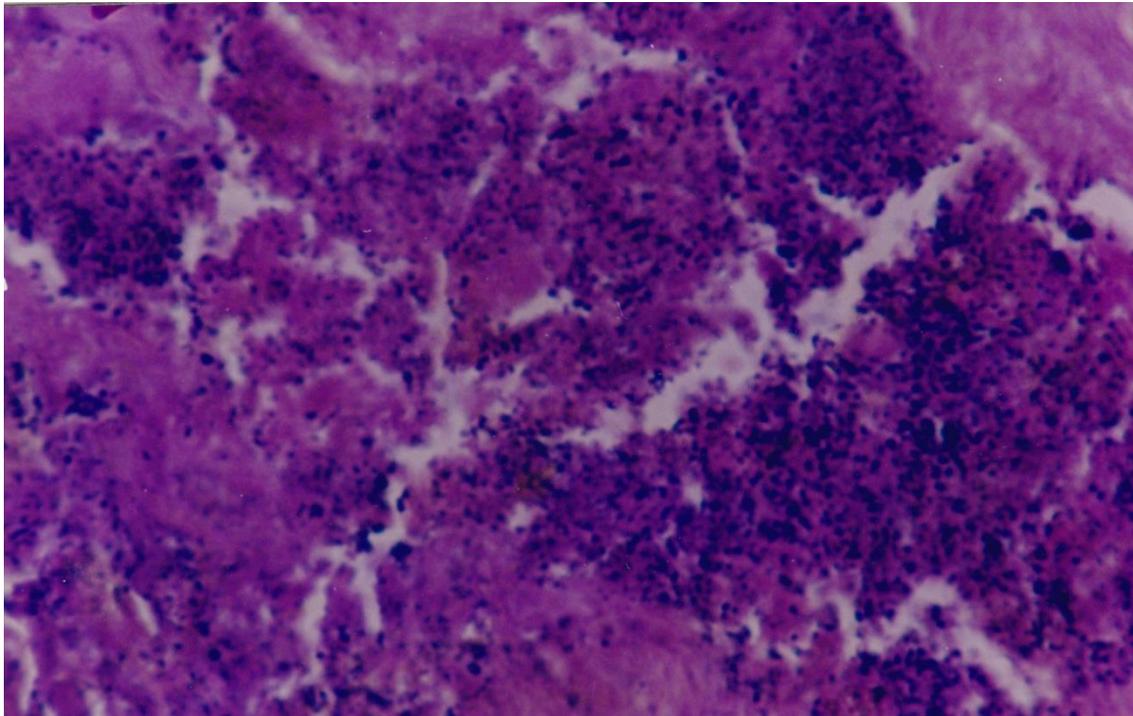


Fig. 12. Portion of liver as in Fig. 11 at higher magnification shows macrophages and lymphocyte. Hepatocyte appeared as homogenize atrophied masses (x 200).

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