

SOME CELLULAR CHANGES IN THE LIVER OF *POMADASYS OLIVACEUS* (DAY) DUE TO NEMATODE LARVAE INFECTION

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ABSTRACT

Pathological changes in the liver of marine fish *Pomadasys olivaceus* (Day) due to nematode larvae is described. Severe destructive effects have been observed resulting into loss of architectural morphology of the hepatocytes, inflammatory host tissue response, various necrotic abnormalities, degenerating bile duct. Clogging of hepatocyte was also observed. Dilated sinusoid is another important observation due to by this infection. Vacuolar degeneration of hepatocytes was prominent.

INTRODUCTION

Pomadasys olivaceus is a popular edible fish commonly called as “Dothar”. It is also a favourable host for various parasites. Several parasites have been reported from *Pomadasys olivaceus* (Akram, 1975; Bilqees, 1976, 1977. Bilqees & Khatoon, 1980; Bilqees & Malik, 1980. Bilqees & Nighat, 1981; Bilqees *et al.*, 2004 and Fischthal & Thomas, 1970). But detailed histopathology of liver of this fish has not been described.

The fishes of Karachin coast are commonly infected with a variety of parasites especially adult and larval nematodes. Nematode larvae of several species are found in the liver of various fishes of Karachi coast. The nematode larvae of anisakid genera *Contracaecum*, *Parrocaecum*, *Anisakis* and *Raphidascaris* are one of the common parasites of the fishes of Karachi coast (Bilqees & Fatima, 1986, 1992; Fatima, 1985; Fatima & Bilqees, 1989).

It is known that nematode larvae affect the growth and health of fishes by destroying the liver and in severe cases death may occur due to these infections. These larvae may affect the fish directly causing traumatic injury, wounds and ulcerations or indirectly by toxins and waste matters produced. The body of fish respond to all types of injuries due to parasite initially by a process of inflammation, with local vascular tissue reaction resulting into leukocytosis, eosinophilia and lymphocytosis. These conditions have been previously reported in the liver of fishes by (Bilqees, 1992, 1995, Bilqees & Fatima, 1992; Bilqees *et al.*, 1993, 1992, 1999).

During the present studies vacuolar degeneration of hepatocytes and disintegration of hepatic cords and lobules was most prominent.

MATERIALS AND METHODS

Fishes were collected from different fish markets of Karachi. Out of 15 specimens of *Pomadasys olivaceus* examined, four were severely infected with nematode larvae and six were lightly infected. Several organs were involved including liver, spleen, intestine, stomach and mesenteries. The liver was more severely affected and selected for the detailed study. For this purpose selected portions of infected liver were fixed in 10% buffer formalin for 24 hours, dehydrated in graded series of alcohols, cleared in Cedarwood oil and xylol and then left in a mixture of equal amount of melted paraffin wax and xylol at 60°C for 12 hours. Then transferred to melted wax and left in an incubator for 6 hours. Wax blocks of the specimens were made in L-blocks by usual procedure. Sections were cut at a thickness of 6-8 microns. Deparaffinized and stained with haematoxylin and eosin by standard method and mounted permanently in DPX. Photographs of selected portions of the sections of infected liver were prepared using Fuji 400 color film on Nikon (Optiphot-2) photomicroscope. Details of damage caused by the nematode larvae in liver of fish are described.

Results

During the present study it was observed that liver was badly damaged by nematodes even if it was physically not present in the area of tissue damage. There was coagulative necrosis and bile duct became fibroid while hyperplasia was also observed in the middle layer (Figs. 1, 2). One of the important finding is the deposition of amyloid between the sinusoids (Figs. 3, 4, 5). In the liver, amyloid is initially deposited in the space of Disse but progressively extends to constrict the hepatic sinusoids and compress the hepatic parenchyma cells. As the condition becomes more advanced as in (Fig. 8). Ribbon like pink staining deposits of amyloid are laid down in the sinusoids. Hepatic cells were compressed and exhibit atrophy. In Figs. 3 and 6, show that vessel wall was thickened by homogeneous pink staining amyloid. Amyloid in small vessels was a subtle feature in many cases. Confirmation of the diagnosis may be made by using a stain such as Congo red.

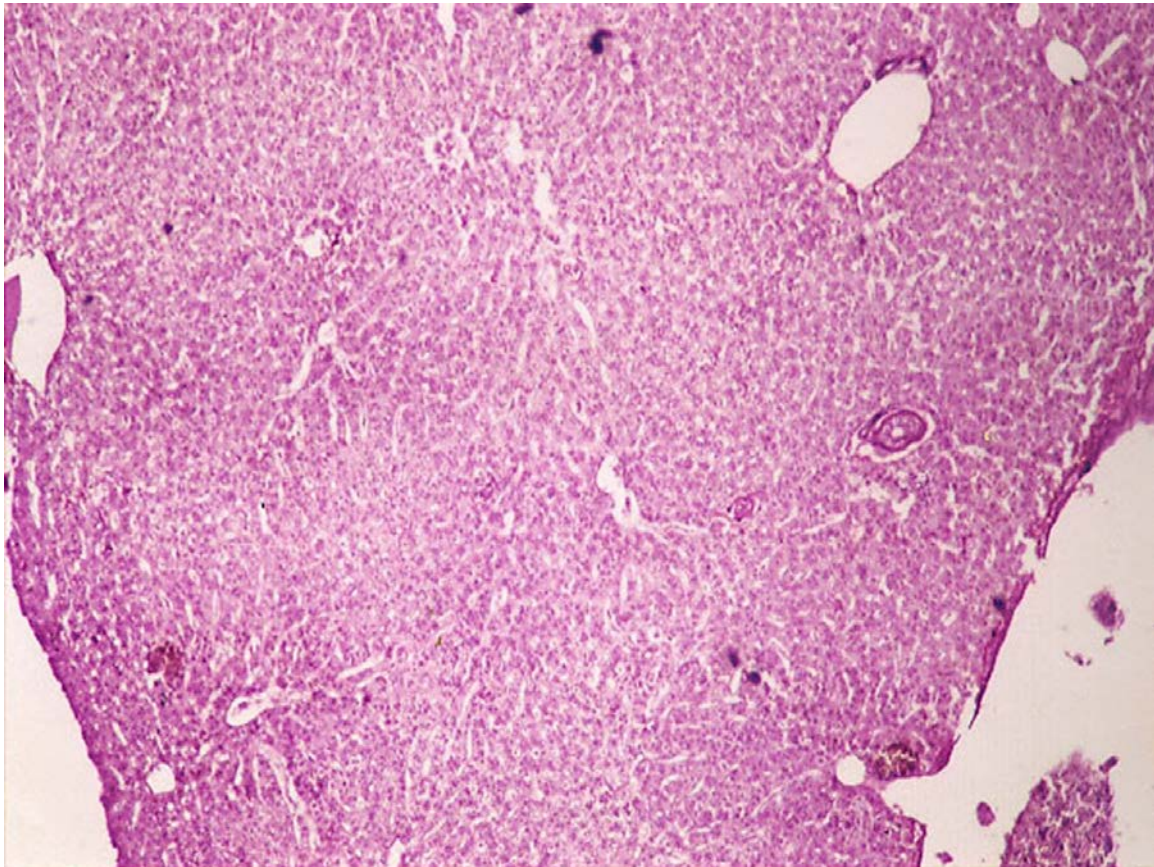


Fig. 1. Portion of liver section showing atrophy and lobular disintegration and degenerated bile duct. X20.

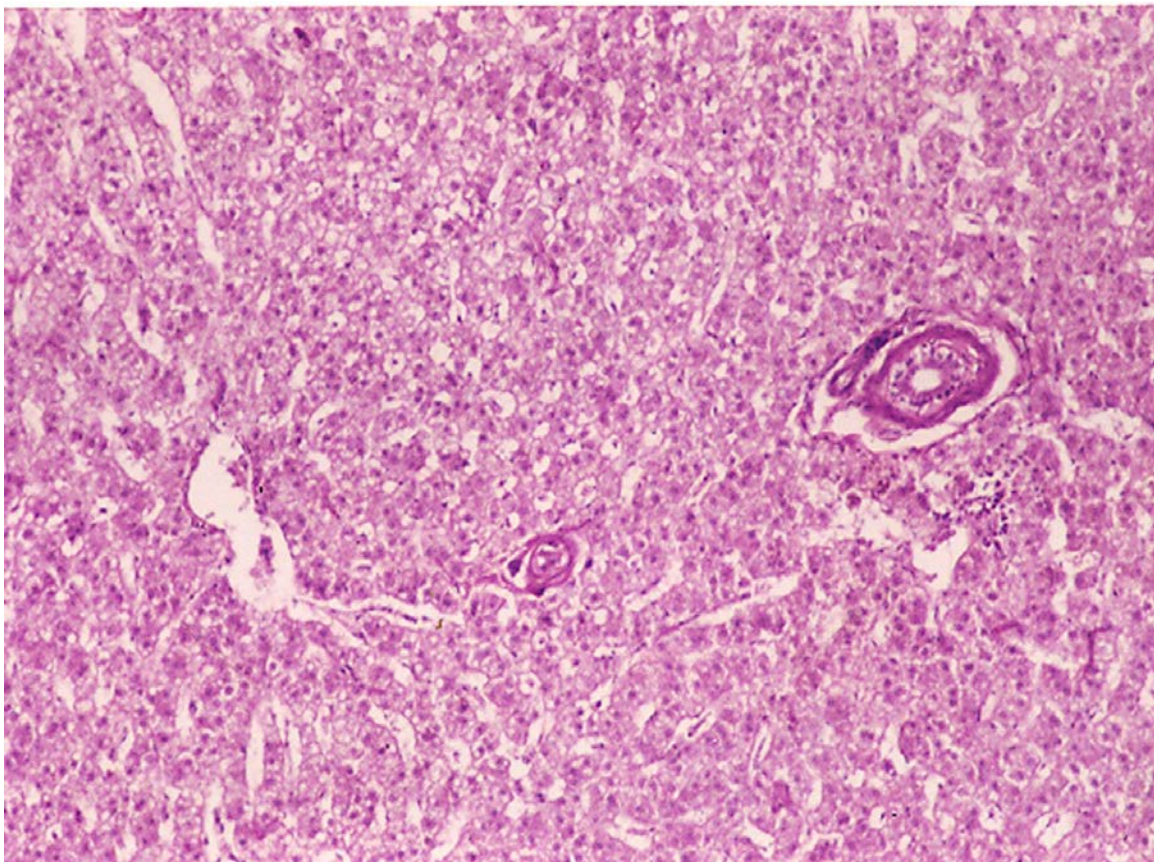


Fig. 2. Portion of Fig. 01 at higher magnification showing degenerating bile duct (arrow), vacuolar degeneration and shrinkage of hepatic cords X50.

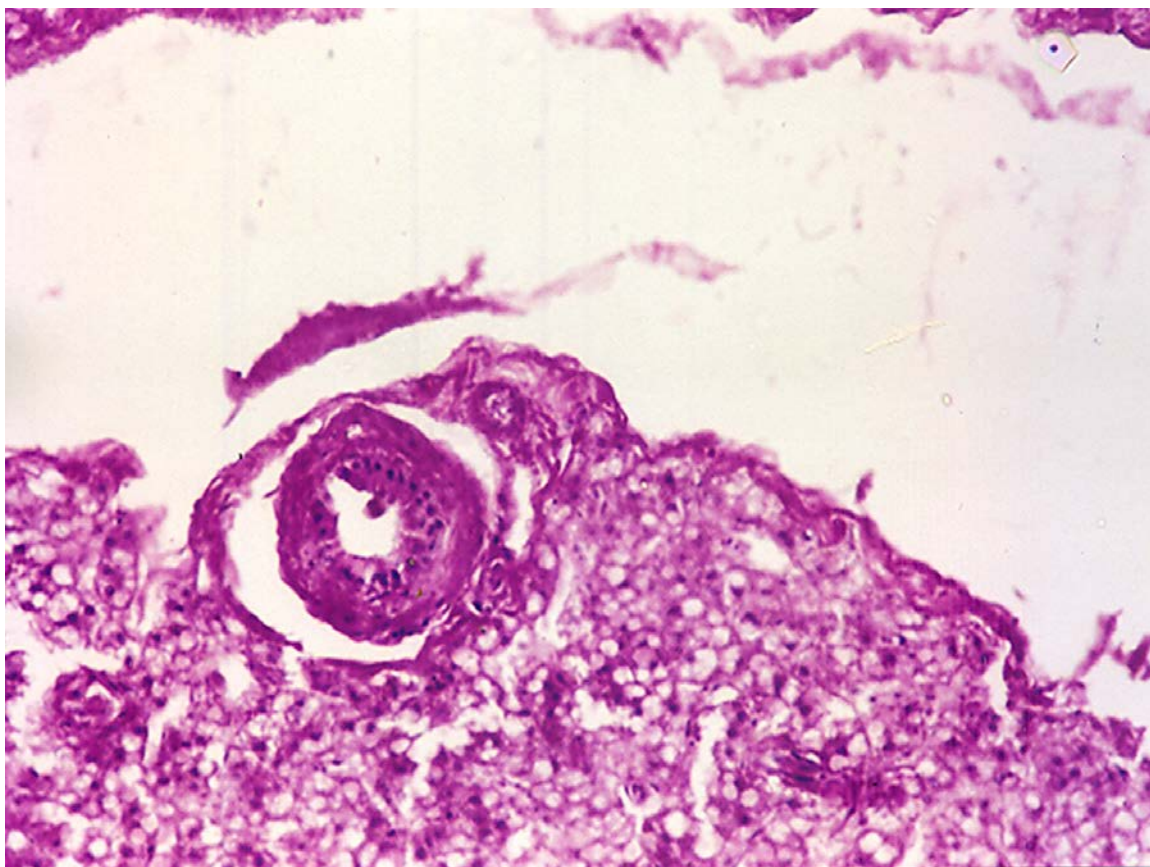


Fig. 3. Portion of liver section shows atrophied and vacuolated hepatocyte and fibrotic bile duct dislocated from the surrounding tissue X100.

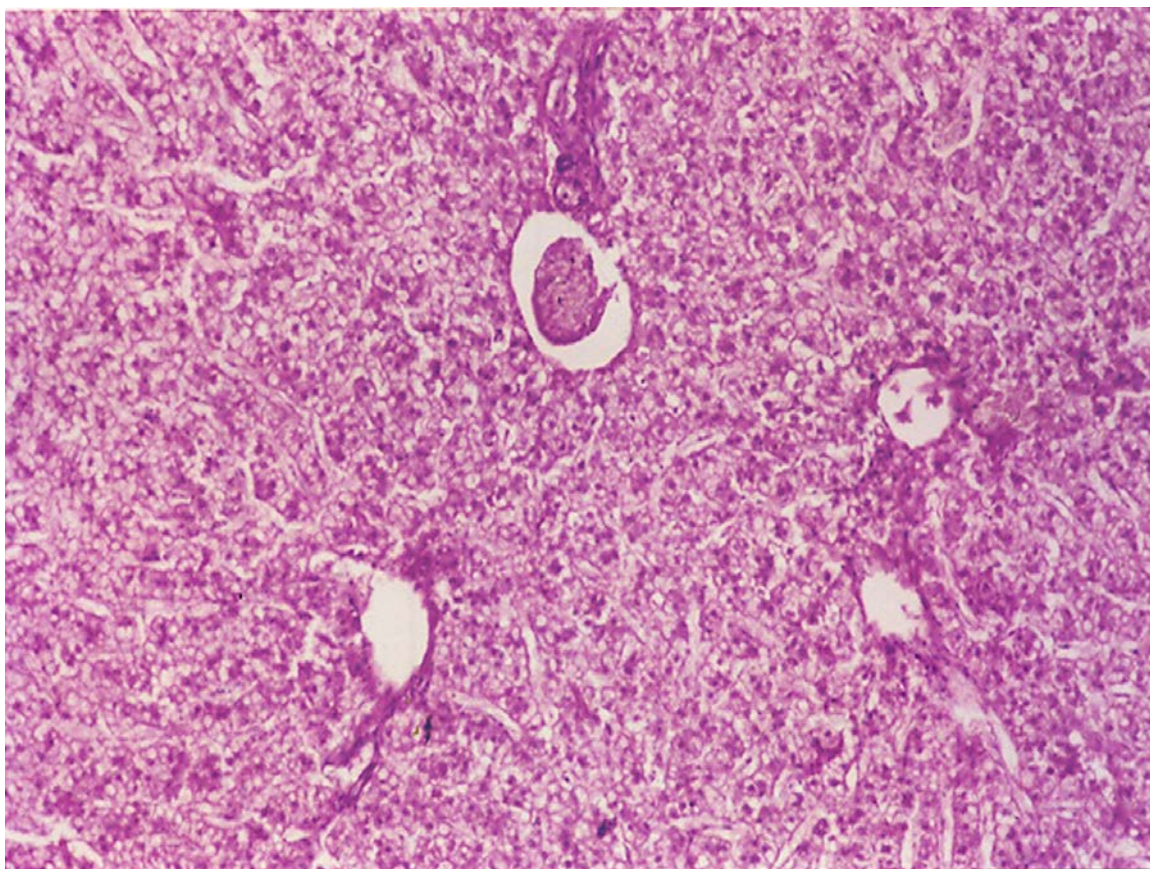


Fig. 4. Portion of liver section shows vacuolar degeneration of hepatocytes and lobular disintegrity. In portal tract area sludging of vein is obvious. Hepatic cells become compressed X50.

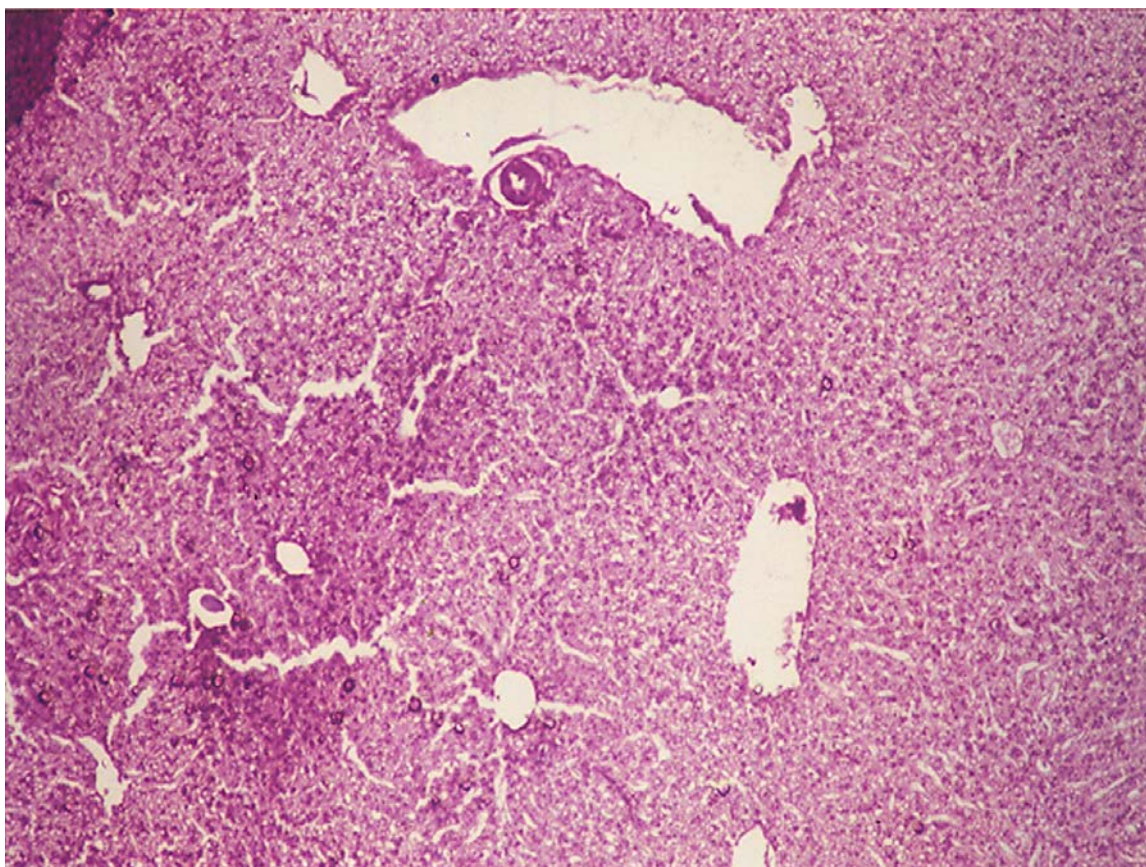


Fig. 5. Liver section shows inflammatory cells infiltration along with and atrophy of lobular disintegration and separation of lobules (arrow) X20.

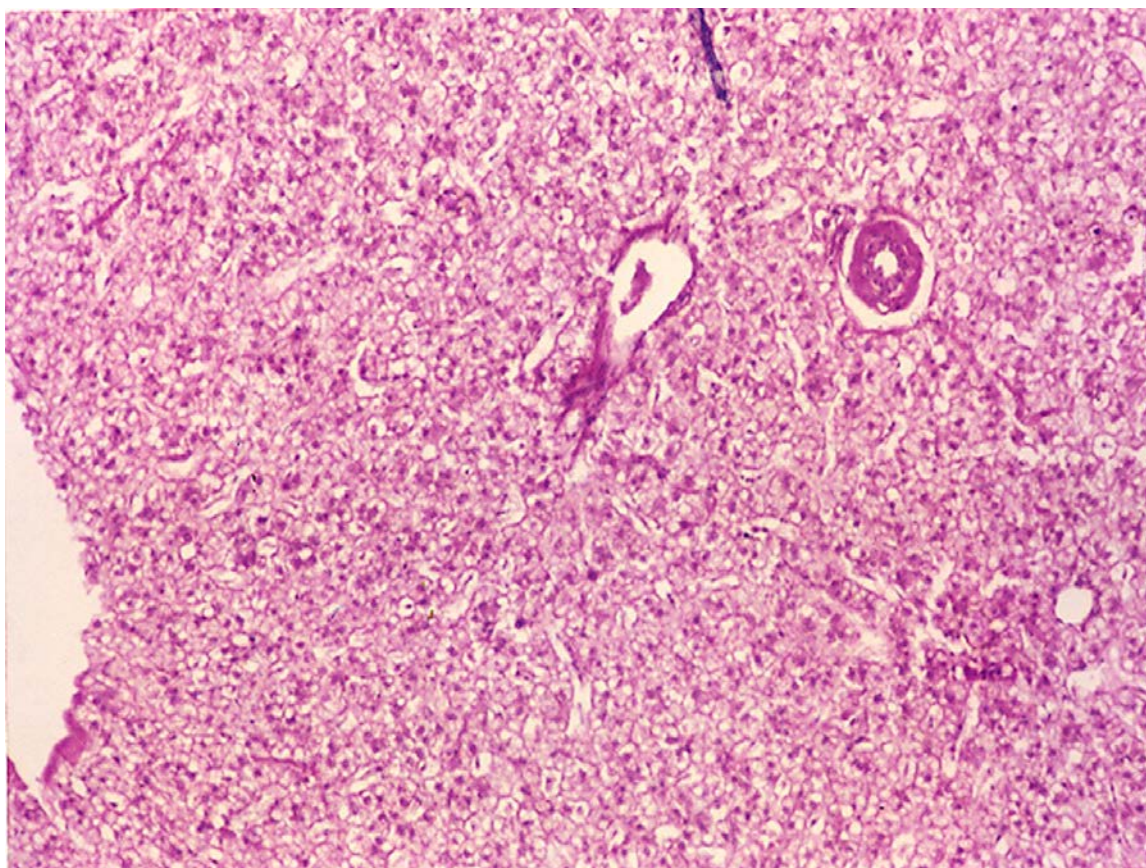


Fig. 6. Liver section showing vacuolar degeneration of hepatocytes and necrosis. Hepatic vessels are also deformed X50.

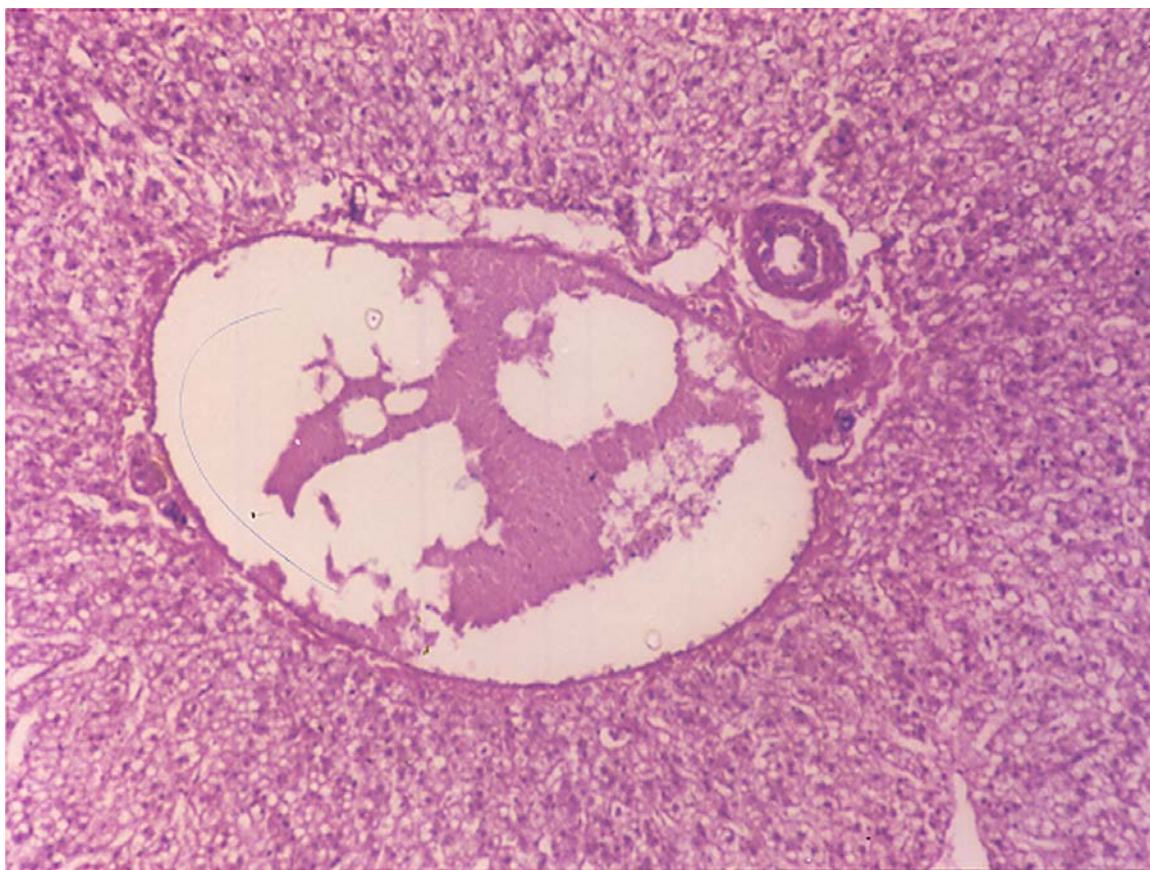


Fig. 7. Section of liver shows atrophied hepatocyte extremely dialated hepatic vein with sludging (arrow) and degenerated bile ducts X50.

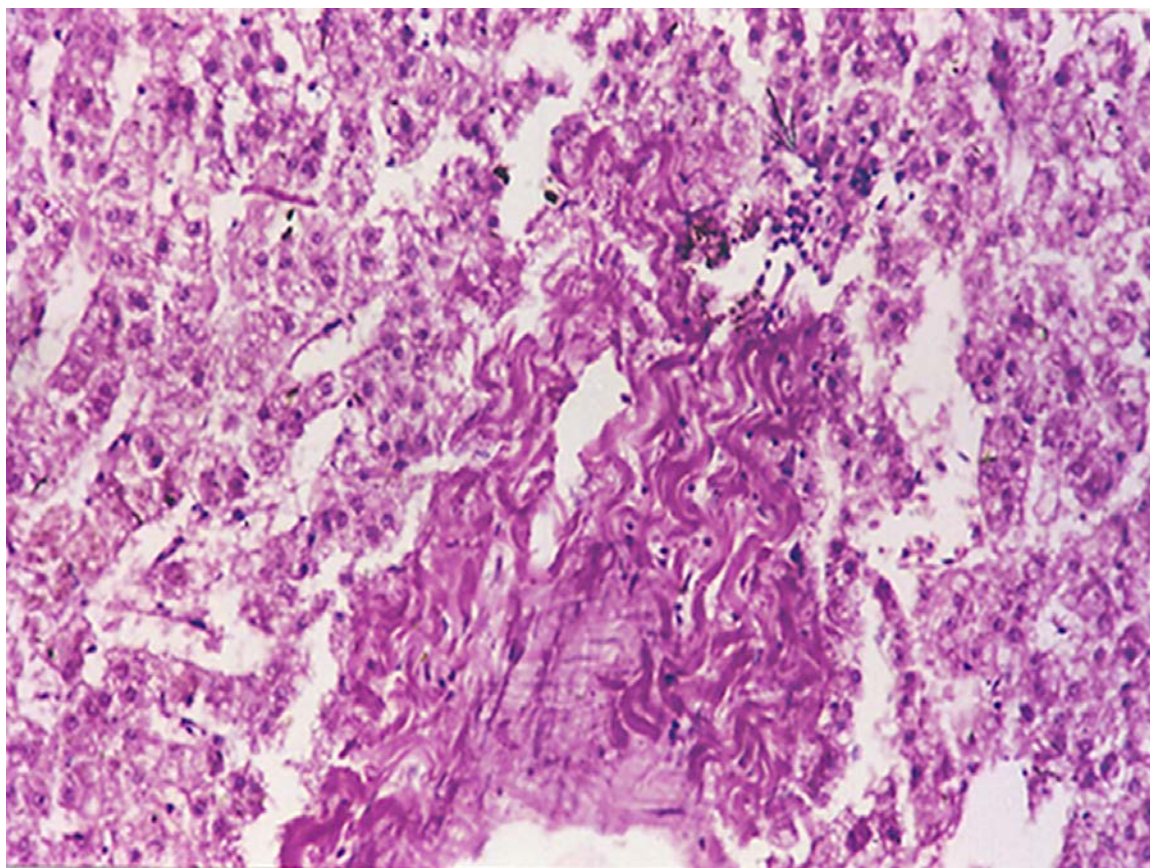


Fig. 8. Portion of liver section shows atrophy of hepatic cells (arrow) necrosis, fibrosis and degeneration. In the center portal tract area is totally damaged X100.

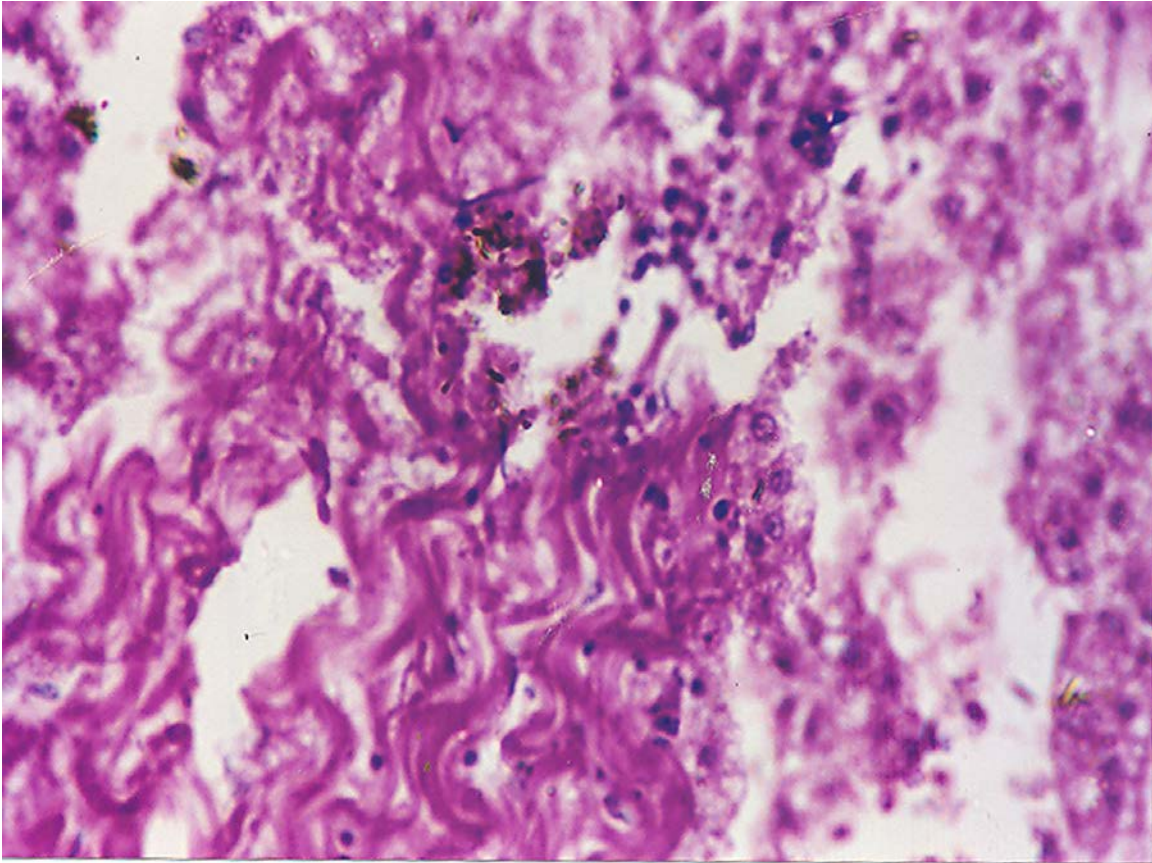


Fig. 9. Section of liver in fig 08 portal tract area at higher magnification showing macrophages, lymphocytes (arrow) at the region of necrosis. Fibrosis is also prominent at necrotic area X200.

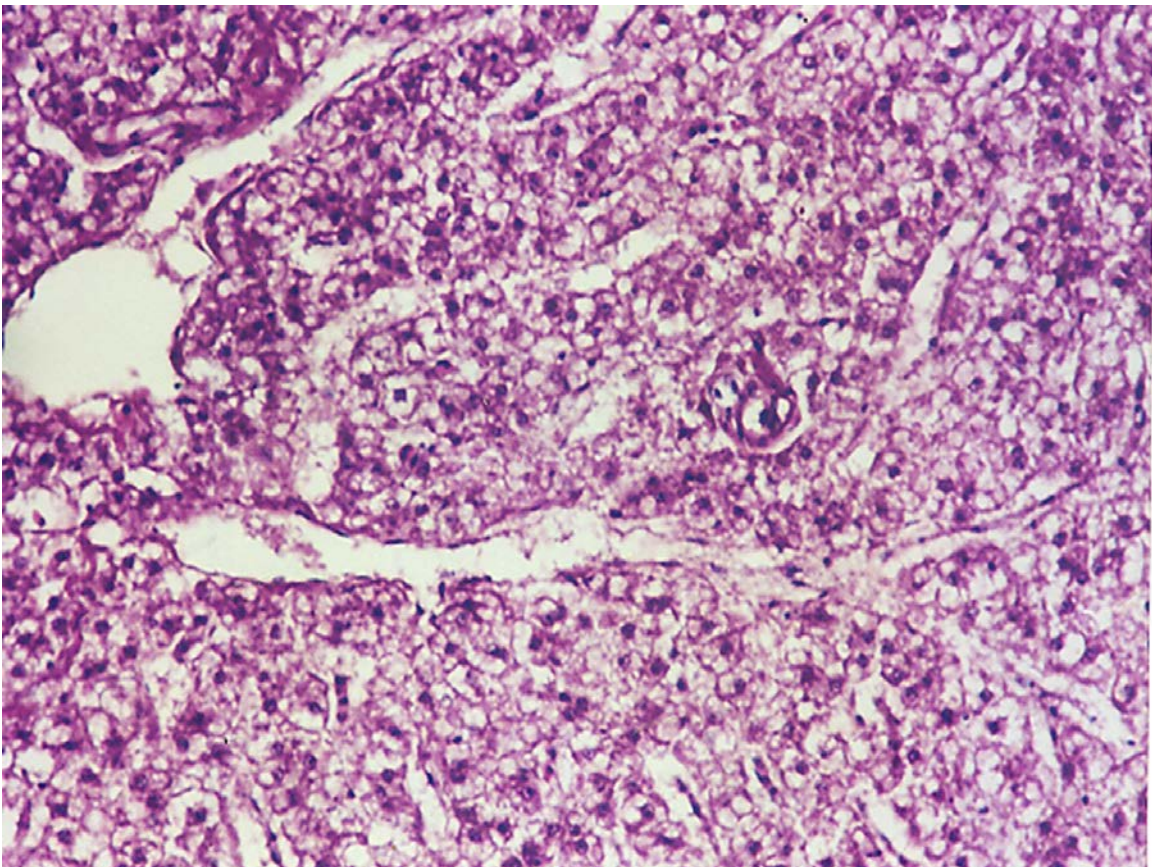


Fig. 10. Section of liver showing vacuolar degeneration of hepatocytes and inflammatory reactions, picemal necrosis in the vicinity of central vein and dilated sinusoids are obvious X100.

In micrograph 8 and 9 note the necrosis and fibrous degeneration, the space in the vascular meshwork vacated by the macrophages are now invaded by fibroblast growing in from the margins of the damaged area. The fibroblast continued to proliferate forming collagen fibers and the capillary network underwent progressive atrophy. There were marked chronic inflammatory changes in the portal tracts which became expanded by chronic inflammatory cells, and the layer of liver cells immediately adjacent to the portal tract underwent necrosis. This type of necrosis was patchy piecemeal necrosis (Figs. 7, 8, 9 and 10).

Discussion

Bullock, 1963 has reported enlargement and inflammation of intestine of salmonid fishes infected with acanthocephala. He reported that total destruction of the epithelium at the point of attachment and paramucosal lumen consisting of large numbers of detached cells of epithelial and connective tissue origin. Thickening of lamina propria was also noted by (Chaicharan & Bullock, 1967). Present observations are basically similar to those reported by Bilqees & Fatima, (1992) and Bullock, (1963) for nematode and acanthocephalan infections. But greater damage and total destruction of mucosal and serosal layers were noted during present investigation. This could be due to the strongly muscular sucker penetration. This might have provoked traumatic severe tissue reaction which resulted into destruction of mucosal layers and underlying tissues.

Capsule formation around the penetrated acanthocephalan and nematode was observed by various workers. No capsule formation was observed around the nematode but muscular degeneration and necrosis was prominent. It was also observed that villi were destroyed to such an extent that they appeared just like a mass of tissues with the penetrated nematode in between. George & Nadakal (1981) reported extensive mucous secretions in the stomach of *Rachycontron canadus* infected with acanthocephalan parasite, but during the present study no mucous secretions was observed due to nematode infestation.

It is concluded that parasite enzymes may be responsible for damages.

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