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BILE DUCT HYPERPLASIA AND FATTY DEGENERATION IN GOAT LIVER INFECTED WITH *FASCIOLA GIGANTICA*

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ABSTRACT

Fasciola gigantica is a common liver parasite in ruminants in Pakistan including goat. This infection is of great economic and public health importance. But the studies on tissue damage caused by this infection are meager specieally in goat. Therefore, observations are made by the study of histological sections of infected liver of goat Microscopic observations revealed severe host tissue reaction and damage including bile duct hyperplasia and fatty degeneration of liver. There was a quantitative increase in the number of cells of bile duct epithelium increasing the volume of the duct. Fatty degeneration indicates severe injury and was obvious by the accumulation of fat vacuoles within parenchymal cells resulting into a foamy appearance under the microscope. Dilated sinusoids, fibrosis, and vascular changes were also observed.

Key-words:- Goat liver, *Fasciola gigantica*, bile duct, hyperplasia, fatty degeneration.

INTRODUCTION

Fascioliasis occurs worldwide and it is of well known veterinary importance. The trematodes *Fasciola hepatica* and *Fasciola gigantica* are the causative agents in America, Europe, Africa, Asia including Pakistan and Australia. It is mainly a parasitic disease of liver and bile ducts of ovines, bovines and other ruminants throughout the world. *F. hepatica* and *F. gigantica* are found in all provinces of Pakistan (Chaudhry and Niaz, 1984; Irfan, 1984; Masud and Majid, 1984; Anwar and Chaudhry, 1984; Durrani *et al*., 1981; Sheikh, 1984; Sheikh *et al*., 2004; Sheikh and Khan, 2000; Bilqees and Alam, 1988, 1991). The adult flukes in the bile ducts cause inflammation, biliary obstruction, destruction of liver tissue and anemia.

During the acute phase of the disease when the immature flukes migrate through the hepatic parenchyma, symptoms like abdominal pain, hepatomegaly, fever, vomiting diarohea, urticaria and eosinophilia are present. In the chronic phase when the adult flukes are within the bile ducts the symptoms are more distinct causing intermittent biliary obstruction and infection are also found rarely such as in intestinal wall, lung, subcutaneous tissue and pharyngeal mucosa.

It is considered that *F. hepatica* infection is more common in ovines (Nwosu and Srivastava, 1993, Schillhorn *et al*., 1980). But during the present study only *F. gigantica* infection was found in goat and not a single *F. hepatica* infection was recorded.

Naturally occurring and experimental *F. gignatica* infection in sheep and goat has been reported previously from Pakistan and other countries (Arora & Lyer, 1973; Hammond, 1973; Mughal *et al*., 1984; Khadim, 1976; Guralp *et al*., 1964). Histopathological observations in goat liver caused by *F. gigantica* are very few as compared to *F. hepatica* in sheep.

The histopathology of goat liver infected with *F. gigantica* has not been studied extensively in Pakistan. Therefore, this project was under taken. The histologic patterns specially bile duct hyperplasia and fatty degeneration are described and discussed with other tissue alterations.

MATERIALS AND MATHODS

Naturally infected liver of goat was used for the preparation of histology sections. Small pieces of liver fixed in 10% formalin were processed for making wax embedded specimen blocks. The sections were cut (4-6 micron), stained with heamatoxylin and eosin by standard technique, photographs of selected portions of sections were taken with Nikon (Optiphot-2) photomicroscope using Agfa colour film.

RESULTS

Studies are based on naturally infected goat liver with *F. gigantica*. Grossly, infected livers appear swollen with yellowish to white raised patches in the area where the parasites were present and tissue were damaged.

Cut surface of infected liver exposed *F. gigantica* in the bile duct several necrotic foci of various sizes appearing as white patches were obvious. Hemorrhages in the liver tissue were also seen. The bile duct grossly appeared thickened (**Fig. 1**).

Histological sections revealed severe tissue damage and alterations in the normal structure of hepatocytes and hepatic cords. The changes were in the parenchyma, portal tracts, vascular channels and bile ducts. Fibrous tracts and hemorrhages were also observed.

Biliary fascioliasis produced necrosis, inflammation, fibrosis, cholangitis and hyperplasia. Fatty degeneration was a prominent feature in the severely affected areas. Lipid droplets, distended sinusoids and degenerating parenchyma was found in several sections. Small lipid droplets were observed in some sections (**Fig. 2**).

In some sections lipid droplets were seen throughout the cytoplasm giving a foamy appearance. Lipid accumulation resulted to coalescense the small droplets into many large vacuoles (**Fig. 3**). The fat cells appeared as clear spaces due to removal of lipid during processing. The surrounding liver tissue also appeared abnormal. Most of the nuclei of parenchyma cells were preserved which were more obvious at higher magnification (**Fig. 4**).

Bile duct hyperplasia with fibrosis and thickening of walls was another abnormality. This thickened bile duct was dislocated from the surrounding tissue leaving small and large spaces. The bile duct epithelium was hyperplastic showing more than one layer of epithelium cells. The surrounding wall was greatly thickened, parenchyma in its vicinity was also atrophic (**Fig. 5**).

The combination of cellular swelling, fatty degenerating and atrophy of liver tissue disrupt the normal hepatic cords and produced a disarray of the lobule. Most of the hepatocytes underwent progressive atrophy and boundry of cells was not distinct.

DISCUSSION

There is a huge amount of literature available on fascioliasis world wide but *F. gigantica* infection is relatively little know than *F. hepatica* in ovines including goat and sheep. Here relevant reports on *F. gigantica* infection and its histopathology specially in goat is reviewed.

In Pakistan there is some work on pathology and histopathology of natural fascioliasis in buffaloes and cattle (Sabri *et al*., 1982 Shaikh *et al*., 2004) and cow (Shaikh *et al*., 2005) but not on goat liver infected with *F. gigantica*. In these and other reports the basic pathology is more or less similar to present observations but the morphological patterns are variable.

Mature *F. gigantica* in the gall bladder of goat causes severe changes. The wall of gall bladder and bile ducts are thickened by epithelial proliferation, fibrosis and mononuclear cell infiltration. The proliferative cholecystitis in experimental goat was similar to adenomatous cholecystitis in cattle naturally infected with fascioliasis (Cheema & Hooshmand–Rad, 1985).

Lesions associated with the migration of immature flukes through the parenchyma are a prominent feature of infection with *F. gigantica.* The size of the hepatic lesions increase during the course of infection with *Fasciala gigantica* and are associated with formation of progressively larger areas of scar tissue in the parenchymal migration tracts as the flukes grow (Wiedosari *et al*., 1991).

In an experimental infection with *F. gigantica* in the lungs of sheep, macroscopically several hyperaemia of 7.5–15.5 mm in diameter were found on the surface of the lung. Juvenile flukes recovered in the lungs were much smaller than those in the liver of the same sheep (Yoshihara *et al*., 1998). It indicates that liver is more suitable for their development. It has been reported that haemoglobin concentration and packed-cell volume also decreases after infection in goat with *F. gigantica* (Haroun *et al*., 1989).

Histopathological changes, which comprised degenerative and necrotic changes in hepatocytes associated with hemorrhage, fibrosis, increased lobulation of the liver, mononuclear cell infiltration with haemosiderin deposition in fluke tracts and portal areas and formation of granuloma around fluke egg and fluke remnants have been described in sheep in Sudan naturally infected with *F. gigantica*. A slight rise in the level of serum bilirublin was also observed with slight hyperglobulinaema and a maked hypoalbuminaemia (Hammond, 1973). It has also been described that *F. gigantica* infection in sheep lung and liver produces lesions characterized by damage to parenchyma (El-Samani *et al*., 1985).

Present observations indicate severe was tissue damage in the liver of goat infected with *F. gigantica*. Massive hepatic atrophy was present. Main findings were bile duct hyperplasia and fatty degeneration or fatty change in addition to several degenerative and necrotic patterns.

Fig. 1. Cut surface of liver of goat infected with *F. gigantica*, bile duct is cut open to show the trematodes (arrow). White patches represent necrotic areas while the dark areas are hemorrhages. Note one swollen bile duct (double arrow).
Fig. 2. Lipid droplets, distended sinusoids and degenerating liver tissue can be seen (X100).
Fig. 3. Showing fatty change. Note the foamy appearance of accumulation of fat vacuoles within parenchymal cells. Surrounding tissue is atrophic and sinusaids are dilated (X20).
Fig. 4. Coalescens of lipid droplets into large vacules. The cells are distended and nuclei are not obvious. Surrounding tissue shows cellular swelling (above) and atrophy (below) (X50).
Fig. 5. Showing section of bile duct with extensive fibrosis of the walls. Bile duct hyperplasia is prominent.

Fatty change in some conditions is indicative of severe injury and may even lead to cell death (Robins and Angell, 1976): Microscopically this change is characterized by the accumulation of fat vacuoles within parenchymal cell. Initially small vacuoles appear throughout the cytoplasm, giving a foamy appearance. In severe

cases lipid accumulation leades to colescense of these small droplets into one or many large vacuoles, the cell is distended and the nucleus is displaced. Similar condition was noted in the present observations on the goat liver infected with *F. gigantica.* The fat cells with haematoxylin and eosin stain appeared as clear spaces due to removal of lipid during processing.

Fatty change in the liver may imply a metabolic over load of cells not affecting function initially. This change is reversible if the causative agent is identified otherwise may produce irreversible injuries to liver which may affect the metabolic activity and growth of animal. This may reduce the quality and quantity of meat and poor skin leather.

Bile duct hyperplasia was another common abnormality found during the present observations. The bile duct was hyperplastic, more than one layer of bile duct epithelium was seen with greatly thickened walls. The nuclei of cells were somewhat larger and hyperchromatic. Bile duct hyperplasia appears to be a common abnormality in *Fasiola gigantica* infection (Shaikh *et al*., 2004). It is also know that migration of immature flukes in the course of ovine fascioliasis causes a colestatic phenomenon responsible for changes in serum and biliary bilirubin levels (Ferre *et al*., 1995). The damage to lung and liver tessiue caused by *F. gigantica* is accompanied by increased activity in serum (El-Samani *et al*., 1985).

There is a great need to carry out extensive research on fasioliasis histopathology and other aspects of the disease. Treatment of animals must be undertaken on a large scale to reduce the economic losses.

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