INT. J. BIOL. BIOTECH., 7 (1-2): 69-72, 2010.

DAMAGE TO PORTAL TRACT AREA IN THE LIVER OF GOAT INFECTED WITH *FASCIOLA GIGANTICA*

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ABSTRACT

Histopathology of liver of goat infected with *Fasciola gigantica* has revealed that portal tract area is greatly damaged. It has become fibrotic affecting the artery, bile duct and portal vein. Fragmentation of bile duct and epithelium sludging of vein was obvious in this area. Diffusion of arterial wall was also prominent. In some cases vein was totally blocked by fibrous tissue accompanied by bile duct hyperplasia. In addition to this hepatic cords and hepatocytes lost the normal shape and architecture.

Keywords: Goat liver, portal tract area, sludging of vein, blockage, fibrosis, fragmentation.

INTRODUCTION

Natural infection of liver by *Fasciola hepatica*, *F. gigantica* and diseases caused by these pathogens are common both in ovines and bovines, in sheep and cattle raising countries including Pakistan (Bilqees & Alam, 1991, 1988; Shaikh *et al*., 2004a-b; Shaikh & Khan, 2000; Chaudhary Niaz, 1984; Nwosu & Srivastava, 1993; Hammond, 1973; Arora & Lyer, 1973; Yoshihara *et al*., 1998; Wiedosari *et al.*, 1991). This infection is of great economic value. Huge literature is available on this subject.

The geographical distribution of the two species of *Fasciola* differs (Mas-coma & Bargues, 1977). *F. hepatica* is believed to be of European origin whereas *F. gigantica* appears to be in Africa and Asia. Fascioliasis is also a public health problem. Human infections have been reported in South America, Europe, Africa, Australia and the Far East. Recent estimates show a worldwide prevalence of 17 million cases and 91.1 million are at risk (Keiser and Utzinger, 2005). Human infection by *F. hepatica* has been declared as an important worldwide health problem (Mas-coma *et al.*, 1999a-b, Mas-coma, 2005). *F. gigantica* infection also occurs in humans. Update and diagnosis on human fascioliacis was also given by Marcos and Terashima (2007).

Relatively little histopathological work has been carried out on infected liver as compared to other aspect of fascioliasis specially in Pakistan.

During the present studies on tissue damage in liver of goat, it was noted that portal tract area was distinctly altered and indicates severe damage. It is described and reported here.

MATERIALS AND METHODS

Liver of goat naturally infected with *F. gigantica* was used for the present study. Histology sections were prepared by cutting small pieces of liver, fixed in 10% formalin. Wax-embedded blocks were prepared by usual method, 4-6 micron thick sections were cut, dewaxed, stained with heamatoxilin and eosin and mounted permanently by standard procedure. Photographs of selected portions of the sections were taken with Nickon (Optiphot-2) photomicroscope using Fuji colour film.

RESULTS AND DESCRIPTION

Present histopathological observations revealed that *F. gigantica* infection in severe cases may cause considerable damage to portal tract area in addition to parenchymal tissue. The main findings were fibrosis and fragmentation of tissue resulting into formation of spaces or vacuoles of various sizes. Bile duct and artery was also affected in this area. Bile duct epithelium was fragmented and its wall was thickened (Figs. 1-2).

In some sections vein was blocked and portal tract area was represented by a thickened bile duct with bile duct epithelium hyperplasia and deposition of thick mass of fibrous tissue. Artery was preserved in this area but with intimal tissue damage (Fig. 3).

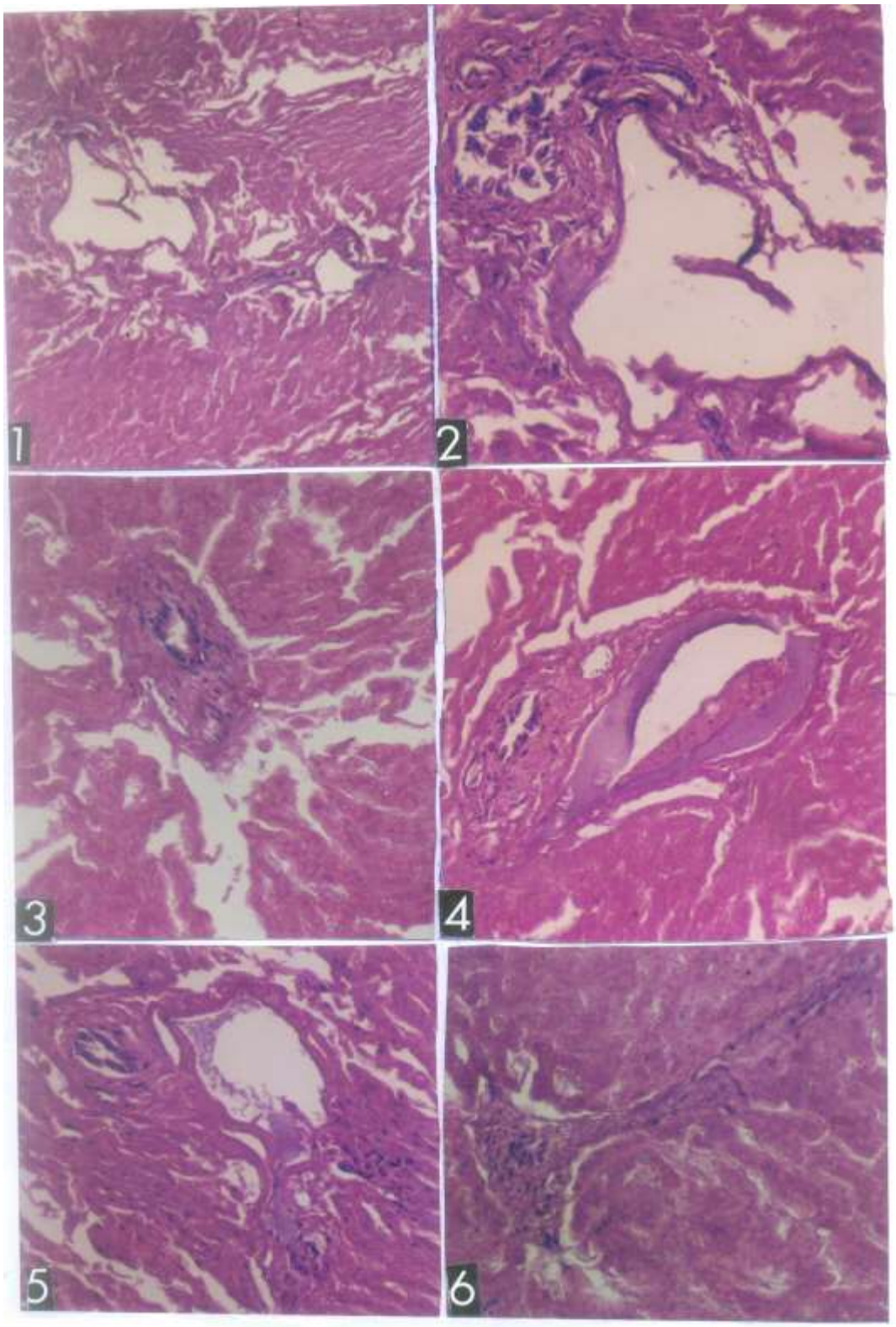

Fig.1-6. Portal tract area in the liver of goat infected with *Fasciola gigantica*.

In some sections damage to veins in portal tract area was more obvious. Vein was thickened due to intimal hyperplasia and deposition of material. Artery and bile duct was preserved but with abnormal morphology (Fig. 4).

Portal tract area shows partly blocked vein and hyperplastic bile duct with liquefactive necrosis of tissue in its vicinity and infiltration of plasma cells and monocytes (Fig. 5).

In some sections portal tract area was totally blocked with blockage of vein by infiltration of fibroblast-like cells and fibrosis. Swelling of parenchymal cells, ballooning and hydropic degeneration of surrounding hepatocytes was obvious (Fig. 6).

DISCUSSION

Liver is a crossroad of the body. The portal and systemic circulation join here to drain through a common venous outflow. The intermediatory metabolism of all foodstuffs occurs in the liver. Hepatic disease does not become manifest until it produces widespread damage. In some severe cases of fascioliasis, disease causes widespread damage and the animals may die.

Fascioliasis usually occur as subclinical infections. These infections cause significant economic losses (Dalton, 1999; Chen and Mott., 1990). Fascioliasis although common in Pakistan, is not identified and cured before slaughtering the animals. The infection may be long standing and may produce irreversible injuries to liver which may affect the metabolic activity and growth of animals.

It is known that *F. gigantica* infection in liver produces lesions characterized by damage to blood vessels in addition to necrosis of parenchyma (*El* Samani *et al.*, 1985). Thickened walls of bile ducts and severe cirrhosis has been reported (Haridy *et al.*, 1999; Shirai *et al.*, 2006). Similar arterial and venous damage was also observed in the portal tract area of goat liver during the present studies. Blood vessel damage was prominent Fibrosis in the portal tract area was also noted. Mononuclear cell infiltration with haemosiderin deposition in fluke tracts and portal areas has been reported earlier (Wescott & Foreyl, 1986).

It appears that artery, vein and bile duct in the portal tract area are damaged. Intensive infiltration of the portal tract areas occur during a secondary liver fluke infection characterized by a pronounced increase in eosinophils, B cells and T cells and the fibroblastic-like cells (Meeusen *et al.*, 1995). Fibroblastic-like cells infiltration in the portal tract areas was also observed during the present studies. Arterial intimal proliferation was also observed as described by Shaikh (2003). Previous studies report that the bile ducts are thickened by epithelial proliferation, fibrosis and mononuclear cell infiltration (Cheema & Hooshmand-Rad, 1985). Similar changes are also noted during the present observations. By the review of literature, it was concluded that portal tract area may be severely damaged due to fascioliasis.

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(*Accepted for publication February 2010*)