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Feline Infectious Peritonitis in a Male Persian Cat

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A B S T R A C T

Feline infectious peritonitis (FIP) is irremediable disease of cats. It is known to be caused by corona virus. During routine necropsy a case of peritonitis was encountered in white Persian cat which was investigated for pathology. Fever, anorexia, jaundice, diarrhoea, weight loss and dyspnoea were the clinical sings reported. Carcass was icteric. Spleen revealed yellowish-white raised nodule scattered over its surface. Liver revealed multifocal necrosis. Kidney was severely congested. Fibrinous exudates were deposited on various visceral organs. Abdominal cavity was filled with yellow, viscous fluid. Histopathological examination was carried out to determine the extent of damage to affected organs. Diagnosis was achieved on basis of clinical signs and pathognomonic histopathological lesions.

Introduction

Feline infectious peritonitis (FIP) is deadly, incurable disease of cats which is reported throughout the world. Though known to be caused by feline corona virus (Hartmann, 2005), it has been complicated by secondary bacterial invaders. Despite of advancement in research till date there is no cure for FIP (Kipar *et al*., 2014). The disease is mainly prevalent in young, purebred and sexually intact male cats (Rohrbach *et al*., 2001). FIP is recorded mostly in pure breeds which might owe to inbreeding of cats (Benetka *et al*., 2004). There are two forms of FIP – wet and dry form. Wet form is characterized by presence of inflammatory exudates into body cavities. Dry form is characterized by its non effusive nature and granulomatous involvement of parenchymatous organs. Although both forms are fatal, the wet form is more common and progressive (Pedersen *et al*., 2009). The present paper we report a case of FIP in Persian male cat with major focus on its clinical features and histopathological diagnosis.

Materials and Methods

Post mortem examination

A carcass of 1 year and 2 months old domesticated Persian intact male cat weighing about 5.5 kg was presented at Department of

Veterinary Pathology, Nagpur Veterinary College, India for post-mortem. Fever, anorexia, jaundice, diarrhoea, weight loss and dyspnoea were the clinical sings reported. Necropsy was conducted with standard procedure.

Histopathological examination

Morbid tissues from these organs were collected in 10% formal saline. These tissues were further processed to form paraffin blocks. Sections of 4 micron were taken and detail histopathological examination was carried out to study the microscopic lesions (Luna, 1968).

Results and Discussion

Clinical signs observed by physician and reported were fever, anorexia, ascities, jaundice, vomition and dyspnoea. Expression of clinical signs depends upon organs affected with granulomatous and Vasculitis lesion (Addie *et al*., 2009). The clinical signs recorded in this study were similar to previous reports (Wolfe *et al*., 1966 and Montali and Stramdberg, 1972). The cat was emaciated and dehydrated. Peritoneal cavity revealed presence of approximately 300 ml of yellow viscous fluid (Fig. 1). All the abdominal organs were covered with yellowish fibrinous material. Spleen revealed yellowish-white raised nodule scattered over its surface (Fig. 2). Liver showed multifocal necrosis along with fibrinous exudates (Fig. 3). Kidneys were severely congested an enlarged (Fig. 4). Gross lesions observed in the present case were also reported earlier by Kipar and Meli (2014).

Microscopic view of granulomatous lesion on spleen showed necrotic centre along with presence of multinucleated giant cell (Fig. 5-7). Whereas germinal centre in spleen had depleted of lymphocytes (Fig. 8). Presence of multinucleated giant cell in spleen is hallmark of the FIP infection in cats. FIP infected cats show apoptosis and depletion of T-cell in lymphoid organs as a result of soluble mediators released at time of infection (Bart *et al*.,1996) which might have contributed for the depletion of lymphocytes. The serosal surface of liver was covered by fibrinous layer along with infiltration of leukocytes (Fig. 9).

Fig.1 Approximately 300ml of fluid

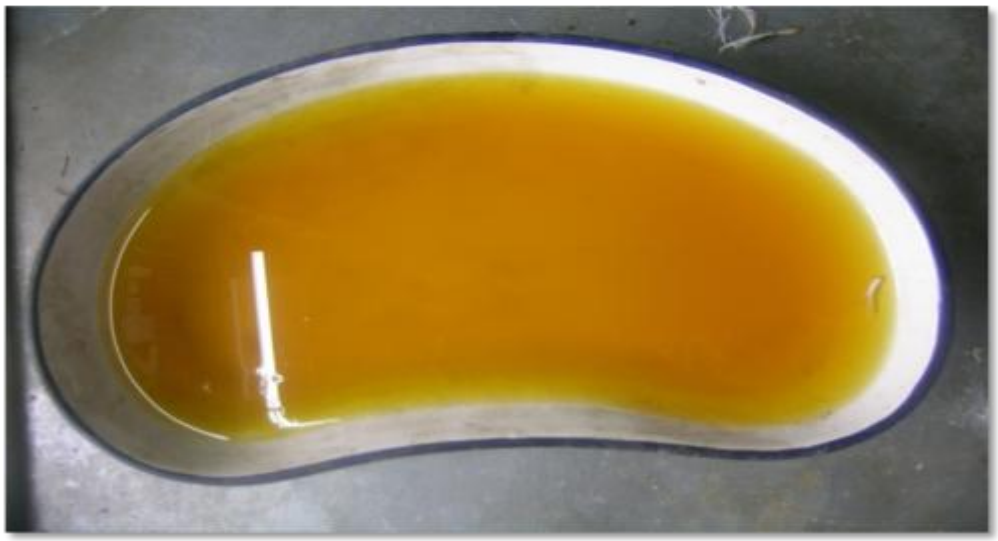


Fig.2 Spleen revealed yellowish-white raised nodule

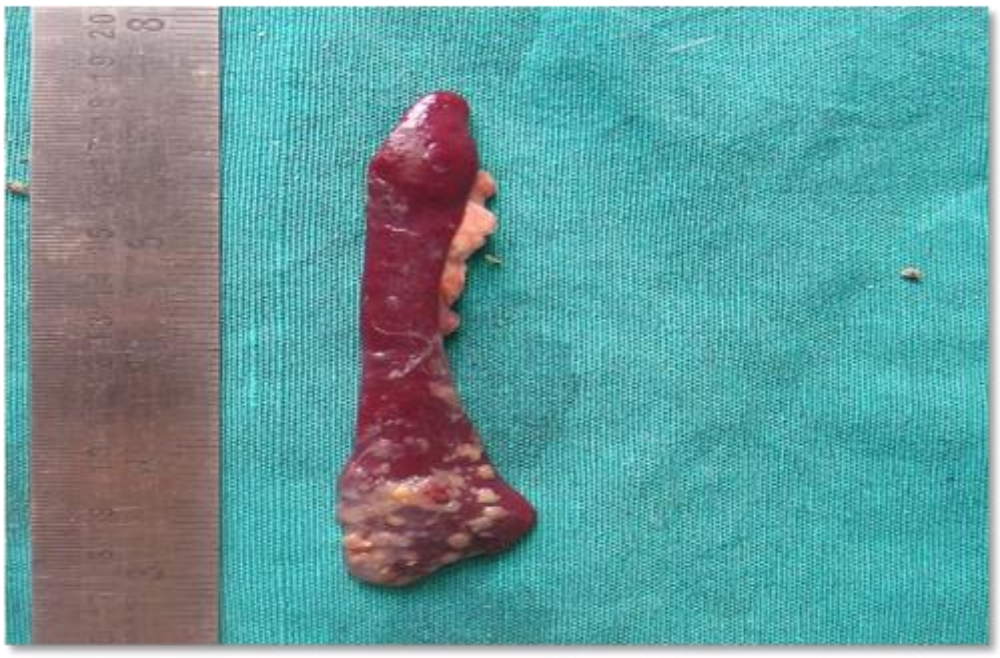


Fig.3 Liver revealed multifocal necrosis scattered over its surface

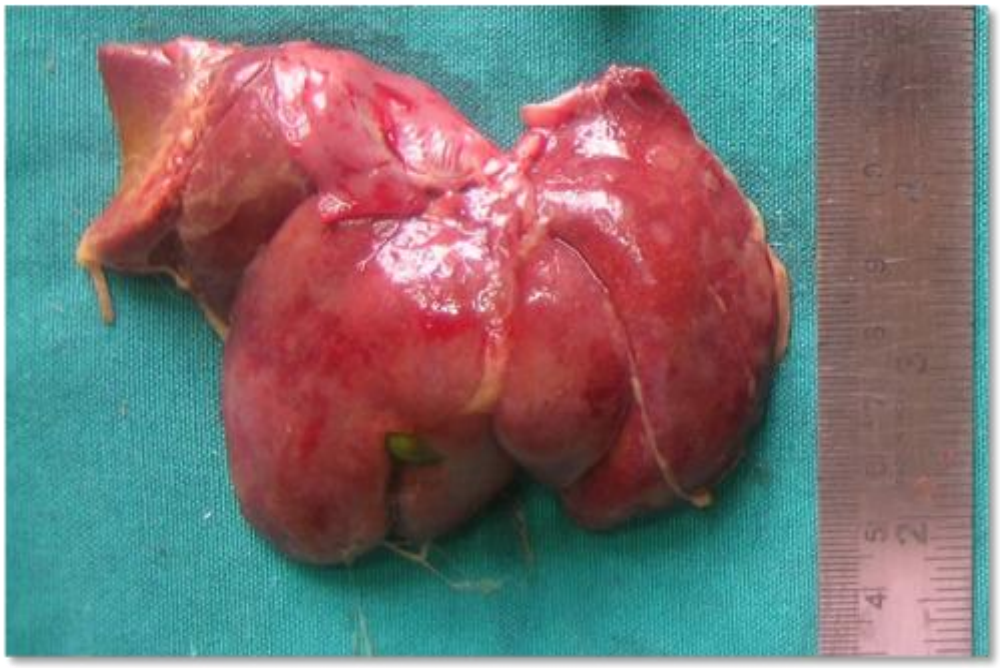


Fig.4 Kidney was severely congested and enlarged

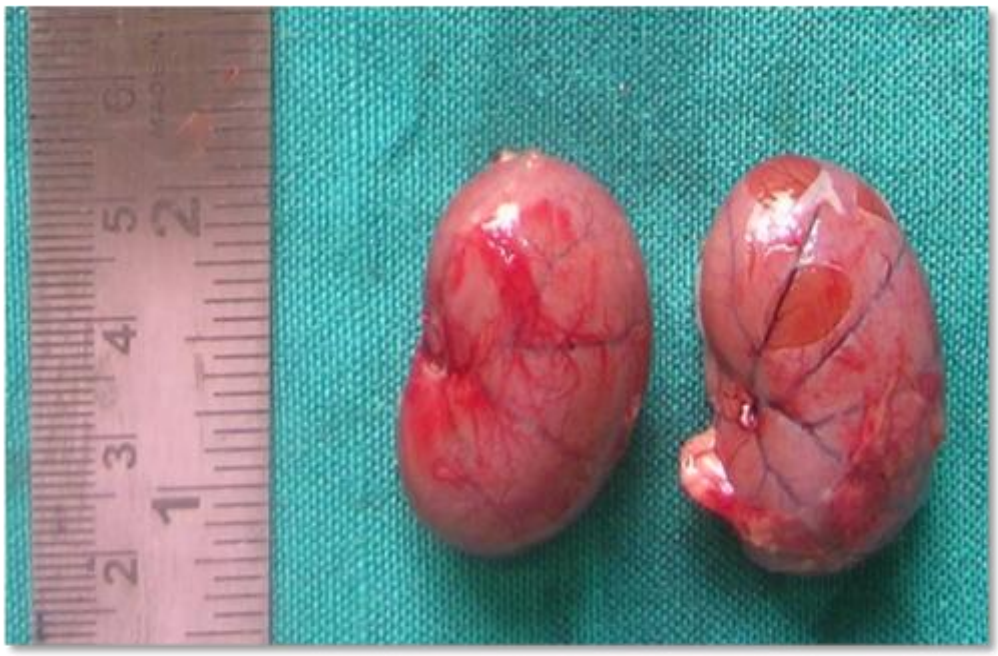


Fig.5 Histological section of nodule on spleen. H&E

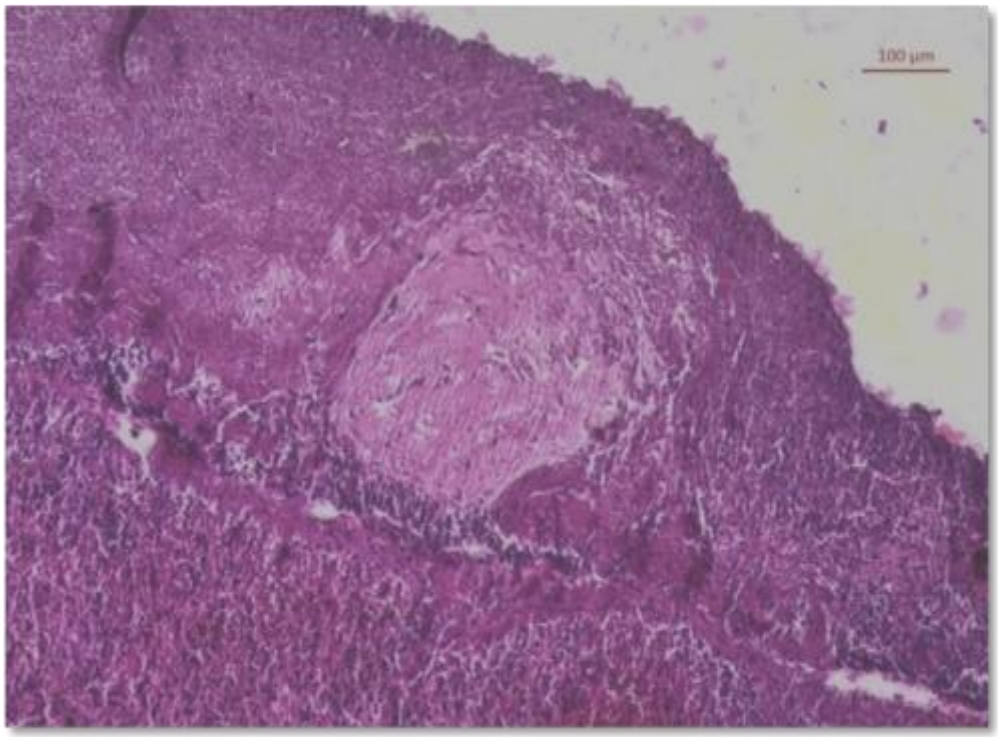


Fig.6 Histological section of nodule on spleen. H&E

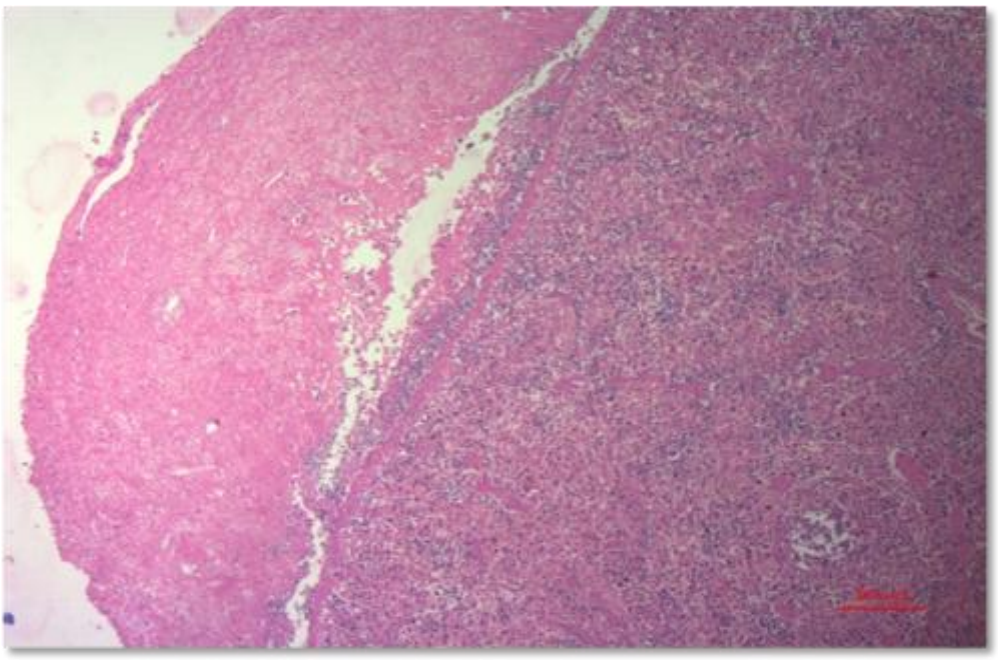


Fig.7 Multinucleated giant cell in nodule of spleen. H&E

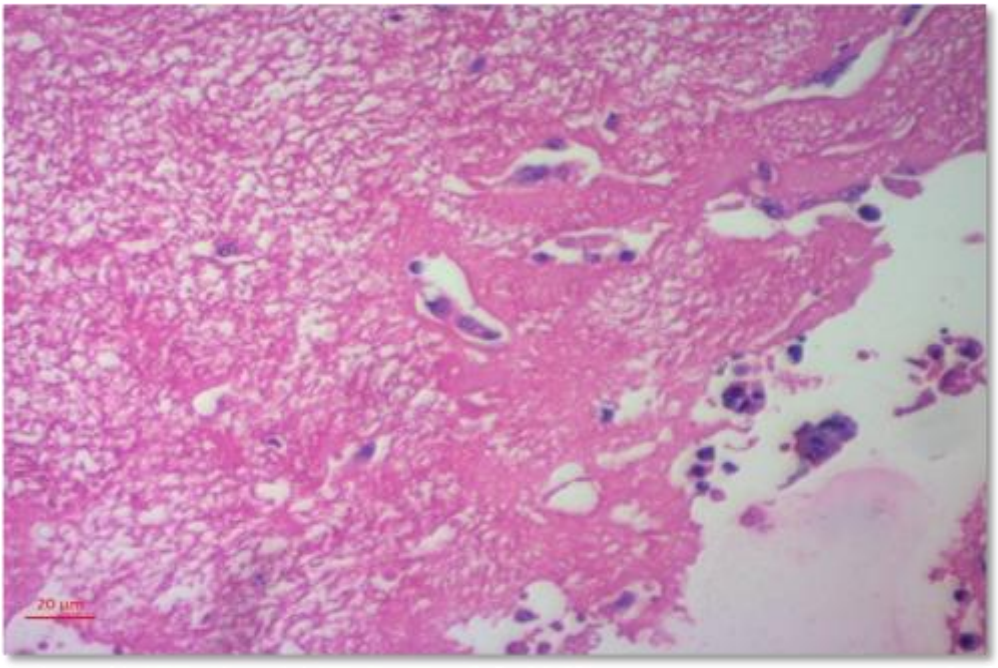


Fig.8 Depletion of lymphocytes from germinal centre in spleen. H&E

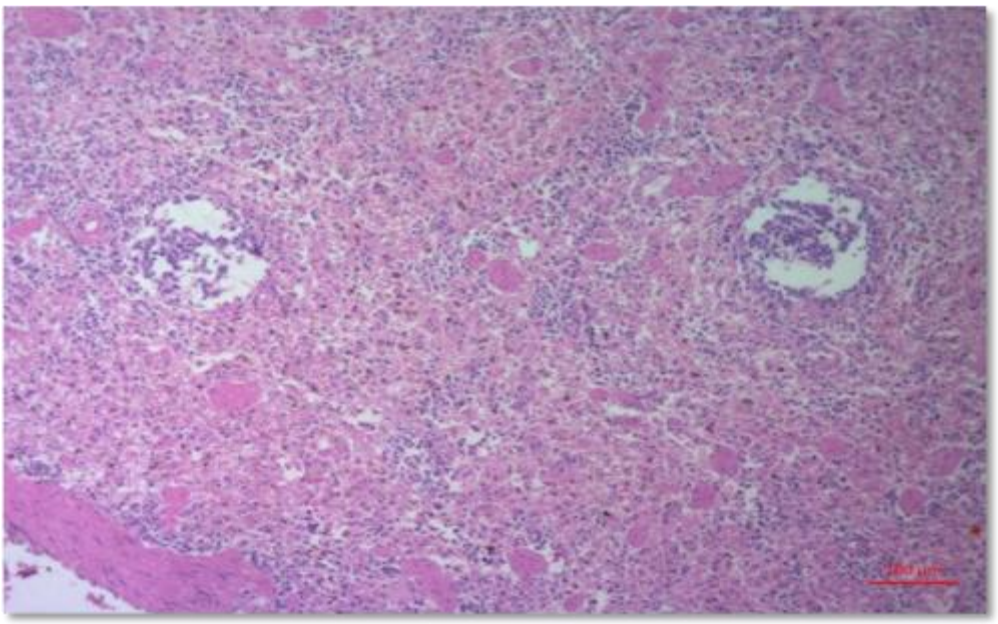


Fig.9 Fibrinous perihepatitis

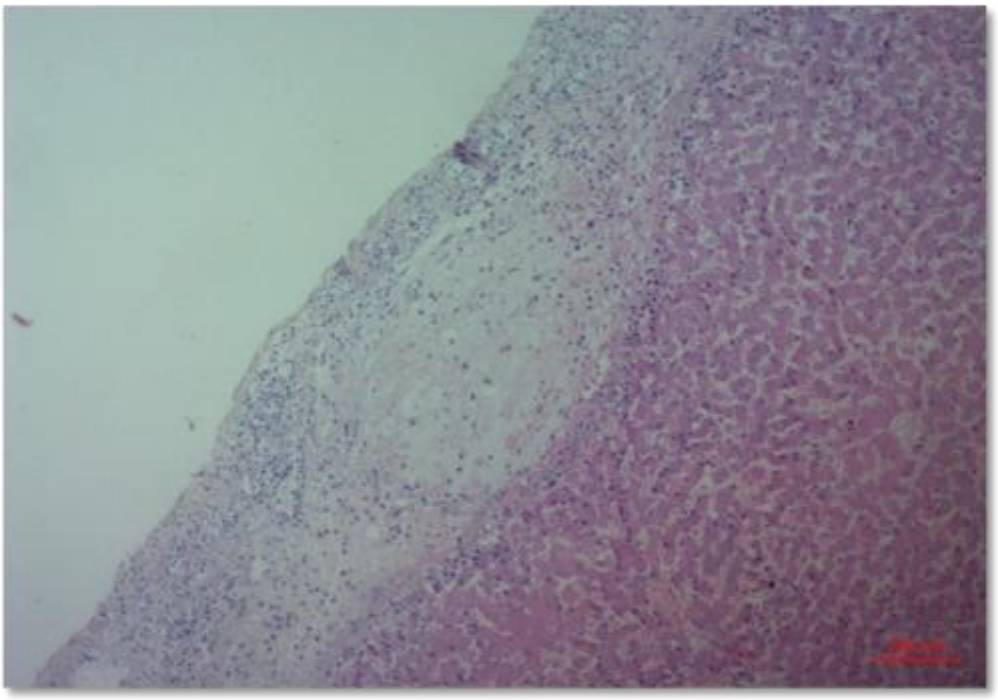


Fig.10 Focal necrosis in liver parenchyma. H&E

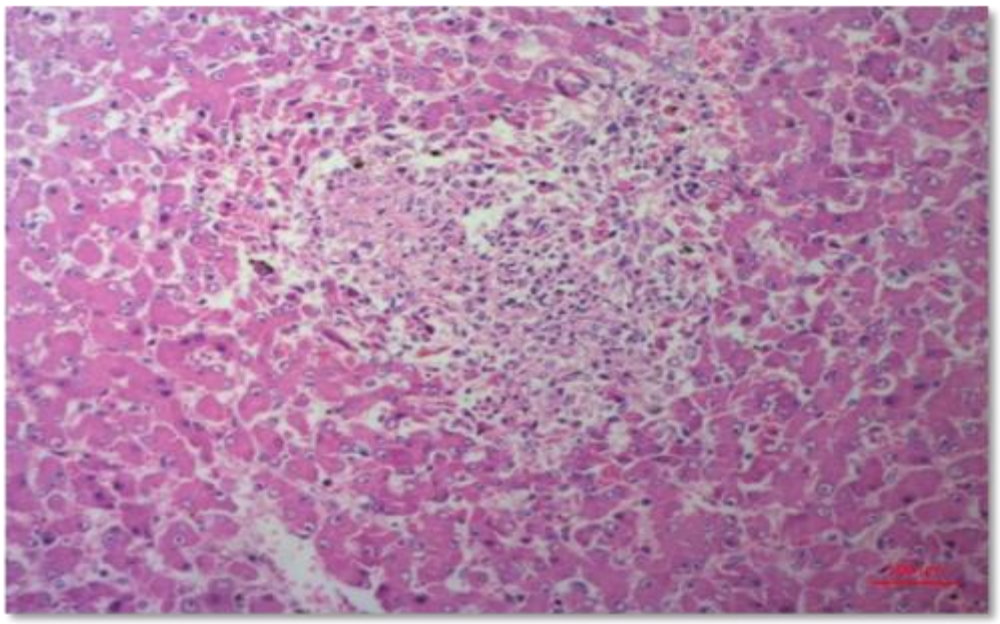
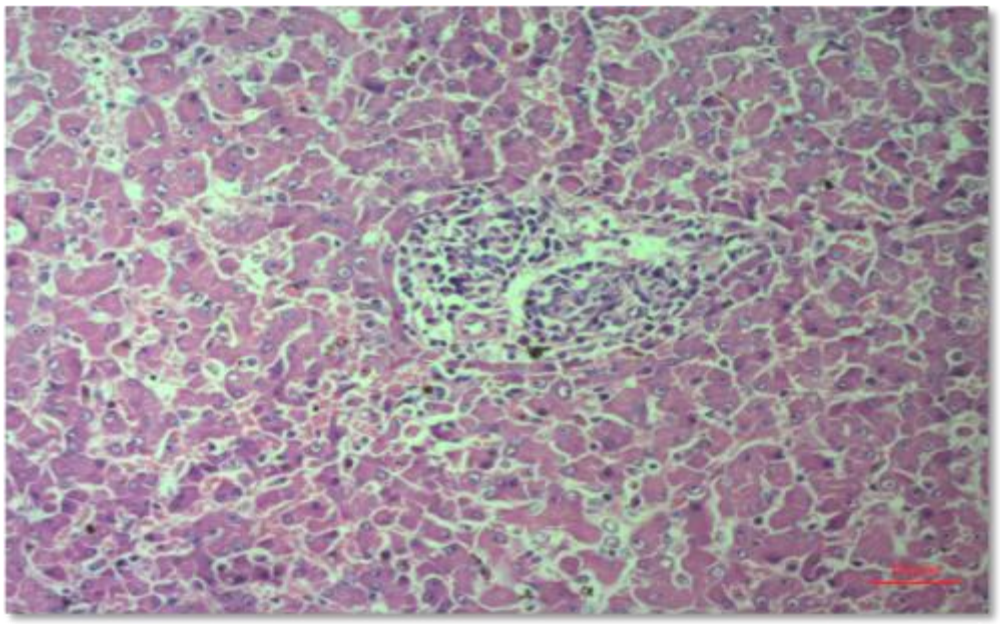


Fig.11 Vasculitis in Liver. H&E



Liver contained multiple distinct foci of coagulative necrosis along with infiltration of leukocyte (Fig. 10). Which often results into hyperbilirubinaemia and icteric condition (Hartmann, 2003). Portal vein in liver showed necrosis and is occluded by leukocytes. Haemorrhages in its surrounding are evident (Fig. 11). Histopathological findings observed in present study were similar earlier observations made by Montali and stramdberg, (1972). Histopathological findings are vital diagnostic tool for FIP, mostly presented with fibrinous, granulomatous and Vasculitis (Norris, 2007).

In conclusion, the anamnesis, gross findings and histopathological findings were most indicative of FIP. Histopathological lesions and extent of damage caused to visceral organs made the young male cat to succumb. FIP is rare and consequences are fatal as there is no effective treatment available. Further research is required to overcome the challenges posed by FIP.

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