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Gross pathological and Histopathological changes in the liver and Bile duct of Sheep with acute and chronic fasciolosis

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ABSTRACT

Fasciola hepatica and Fasciola gigantica are the most widespread liver flukes found in the liver and bile duct of Ovines and Bovines, responsible for major economic losses worldwide mostly by reduction in weight, draught-tolerating capacity, fertility and lactation. In the present study, we tried to examine the changes in the gross morphology and histology of liver samples isolated from local sheep breeds of Kashmir valley, by infection with fasciolosis. For this, Liver samples were collected from slaughtered sheep of different age groups from different regions of Kashmir valley. The gross pathological and histopathological changes were recorded for Fasciola infected livers which were then compared with the livers from the normal sheep. Liver gross pathology in case of fasciolosis showed enlargement of the organ (hepatomegaly) and hemorrhages. Liver parenchyma showed necrosis and fibrosis. Whereas the gross pathology of bile duct showed engorgement of bile in the duct and observation of blackish brown exudates in the bile duct. The histopathological changes in sheep liver by fasciolosis were characterized by atrophy, necrosis and infiltration of fibroblasts and mononuclear leukocytes along the migratory tract. Portal fibrosis, individualization of the hepatic cells and giant cell formation was also observed in the hepatic tissue. Histopathological sections of bile ducts showed fibrosis, fibrous cholangitis and Portal triaditis.

Key words: Fasciola gigantica, Fasciola hepatica, Fasciolosis, Hepatomegaly, Hemorrhages, Liver parenchyma, Atrophy, Necrosis.

1. INTRODUCTION

Parasitic diseases are the major impediment in growth and development of livestock health [1]. One of such economically important parasitic disease is the fascioliasis which is caused by fluke infestation in the liver and bile duct of sheep and cattle mostly by two species of genus Fasciola i.e *Fasciola hepatica* and *Fasciola gigantica*. It has been reported that sheep are more susceptible to the fasciolosis than cattle. Moreover other animals like horses, deer, goats and even humans may get infected with these liver flukes [2]. The global economic loss caused by fasciolosis infection is estimated to be about US$ 3.2 billion annually which may be due to decline in the total body weight gain (low meat yield), draught capacity, reproductive potential and milk production [3,4]. High prevalence is reported from the areas which are surrounded by inland water bodies such as dams or ponds in which snails, particularly of genus Lymnaea are found [5].

The liver is one of the most vital organs in the animal's body. It plays a key role in the metabolism of many endogenous and exogenous substances and as a result, is one of the frequently affected organs in a diseased body [6,7]. Any disease of liver can prove fatal for the animal owing to its imperative role in the body. The root cause of liver condemnation through post-mortem examination are diseases caused by parasites mainly *Fasciola spp*., bacteria and viruses [8-10]. In the fasciolosis infected animals, the damage to the host species is either due to the mechanical or chemical effects by these parasites or by the hosts inflammatory and immune responses. The mechanical injury usually occurs when the infective metacercariae migrate through the liver capsule and hepatic tissue. This migration in turn is associated with trauma, hemorrhages and necrosis of the liver tissue, followed by subsequent granulation which ultimately leads to liver cirrhosis [11]. The other pathological features in case of chronic fasciolosis are the development of hepatic fibrosis and thickening of the bile ducts. The chemical effects involve a strong Th2 response, which in turn is associated with the production of cytokines thereby contributing to the overall pathophysiological condition. However the extent of the clinical symptoms and the lesions caused by these parasites are also related to number of ingested metacercariae, host species affected, duration of challenge, nutritional status and metabolic demands.

The gross pathological changes of the liver in case of fasciolosis infected animals are generally characterized by having an asymmetrical outline, being pale and stiff. The ventral lobe has been found to be most frequently affected and reduced in size. The liver pathology in case of chronic fasciolosis is characterized by hepatic fibrosis and hyperplastic cholangitis. The occurrence of fluke eggs in the hepatic tissue or bile ducts may occasionally stimulate a granuloma like reaction which as a consequence may obliterate the function of affected bile ducts. Calcification of bile ducts, gallbladder enlargement and anomalous migration of the flukes is also common in infected animals [12]. Most studies related to the pathogenesis of fascioliasis focus on the hepatic changes. Reports of injuries in other organs and tissues are quite uncommon [13], and therefore, it is indispensable for a suitable laboratory model to

consent the complete investigation of *F. hepatica* pathogenesis as it migrates through the thoracic and abdominal cavities before piercing the liver.

The development of infection in definitive host is divided into two phases; migratory phase and the biliary phase [14]. The migratory phase also called the parenchyma phase begins when the juvenile liver flukes penetrate the intestinal wall. After penetrating through the intestinal wall, flukes begin to migrate through the abdominal cavity and finally make way into the liver, resulting in the formation of lesions. *F. hepatica* has a strong proclivity for the liver tissue and cause severe intensity of liver lesions. The second phase (the biliary phase) begins when flukes penetrate the biliary ducts of the liver, where they mature, feed on blood and bile and produce eggs [15]. Clinically Fascioliasis is manifested by dullness, loss of appetite, weakness and oedematous distension of mucous surface of conjunctiva and pain on pressure exerted over liver in acute phase of infection. Thus the aim of the current study was to detect the gross and histopathological lesions in the sheep livers and bile ducts due to Fascioliasis in local sheep breeds of Kashmir Valley.

Materials and methods

1. Collection of samples

Seventy five liver samples were collected randomly from slaughtered sheep of different age groups and sexes from different areas of Kashmir valley during different periods of the year.

2. Examination of liver, gall bladder and collection of parasites

In the laboratory the livers and gall bladders were subjected to thorough investigation for the collection of parasites as well as for pathological studies. The gross pathological changes were recorded carefully. The bile ducts were opened first for chronic fasciolosis. For generalized liver fluke infection (fasciolosis) incision was given in different parts of the liver to examine the presence of fluke in the parenchyma. The liver was cut into slices of 4-5 mm. thickness using a sharp knife and pressed to squeeze out flukes from its tissue and smaller bile ducts. Normal saline was used for quick removal of flukes from the liver tissue.

3. Tissue preparation for Histopathological examination

The next step was to study the histopathology of the infected liver tissue which was done by using the methodology of [16]. For this the liver sample was cut into small pieces of about 4-5mm thickness and immediately fixed in 10% formalin. The fixed specimens were trimmed, washed and dehydrated in ascending grades of alcohol, cleared in xylene and finally embedded in paraffin. The embedded samples were sectioned at 3-5 micrometre thickness using a rotary microtome. All the sections were stained routinely with Haematoxilin and Eosin for detailed histopathological examinations. The slides were then carefully observed under microscope (Olympus) for accurate interpretation of results.

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RESULTS

a) Gross pathology:

Liver gross pathology in case of fasciolosis showed increase in the size of the organ (hepatomegaly) (Fig:1) due to inflammatory changes in the parenchyma and hemorrhages (Fig:2) on the parietal surface of the liver, paleness in some areas which was due to the necrotic or damaged region, congestion, firm whitish areas within parenchyma regarded as fibrosis and abscess with calcification in few cases (Fig: 3). Gross pathological changes in bile duct included engorgement of bile in the duct (Fig:4). By cut section, the swollen and the fibrotic bile ducts were clearly visible and blocked by twisted flukes. The bile ducts were found filled with blackish brown exudates (Fig: 5).

Fig 1. Hepatomegaly of sheep liver

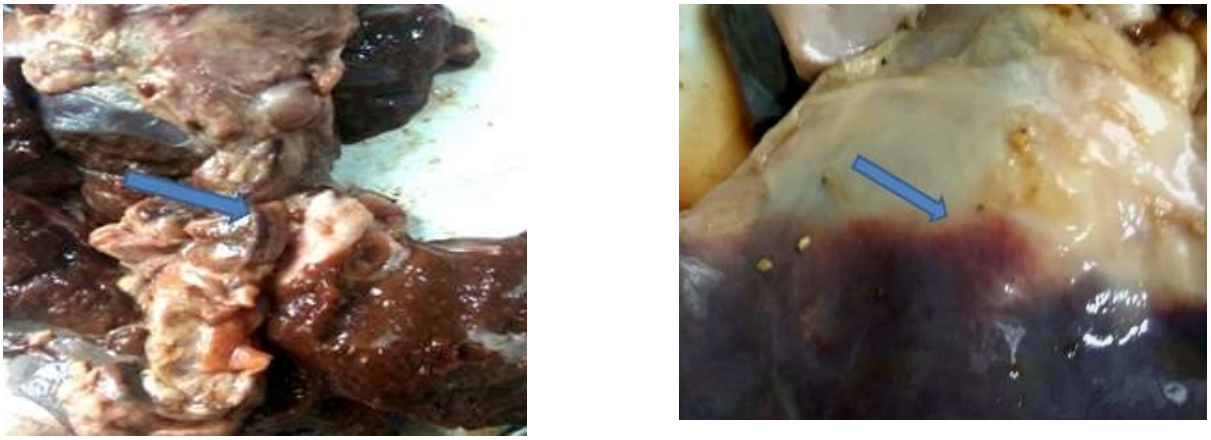

Fig 2. Sheep liver with advanced immature fluke infection showing hemorrhages.

Fig 3. Sheep liver showing Necrosis and Fibrosis

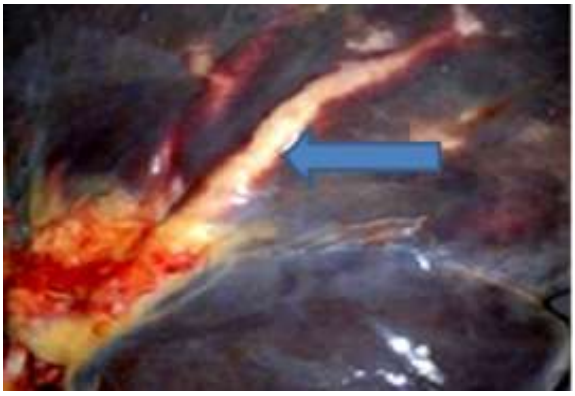

Fig 4. Engorgement of bile duct

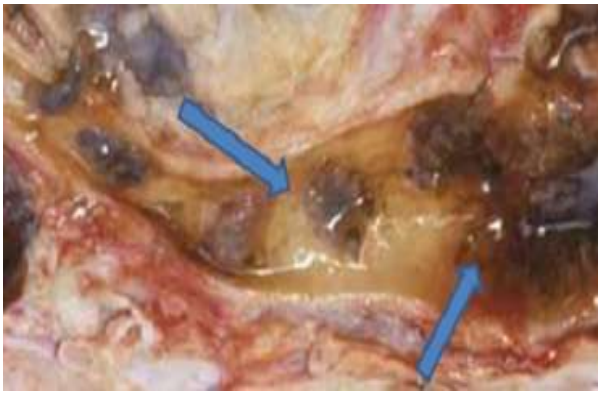

Fig 5. Bile duct of sheep filled with blackish brown exudates infected with adult flukes.

b) Histopathological Examination

Microscopically, contrary to the normal structures in the uninfected liver (Fig: 6), various histopathological changes were observed in the liver and bile duct sections of *Fasciola* infected sheep, which occurred in varying degrees depending on the intensity of the infection. The histopathological changes in sheep liver in chronic fasciolosis were characterized by infiltration of fibroblasts and mononuclear leukocytes (chronic inflammation) in the area previously migrated by young flukes. The hepatocytes showed fatty change in which clear vacuoles appeared in the cytoplasm with peripherally located nuclei (Fig: 7). Liver section also showed atrophy and necrosis of hepatocytes due to chronic Fasciolosis (Fig: 8). The parasitic migration through the liver and parenchyma tissue had resulted in serious damage to the hepatocytes. Their cell walls were degenerated, the nuclei deformed and the cytoplasmic contents emptied into the sinusoids resulted in vacuolar degeneration of hepatocytes (Fig: 9). This indicates the acute phase or the parenchymal phase of the infection with its consequential pathological effects. Some sections showed hepatocytes ballooning degeneration with periportal vein cellular infiltration of mainly eosinophiles, macrophages and lymphocytes (Fig: 10). Portal fibrosis characterized by extensive fibrous connective tissue proliferation in the portal area with infiltration of mononuclear inflammatory cells**,** compression atrophy of hepatocytes adjacent to

fibrosis zone was recorded. In some cases it was accompanied by multinucleated giant cell formation (Fig: 11). Some sections were characterized by distortions and individualization of the hepatic cells (Fig: 12). Some sections also revealed the hyperplasia of the bile ducts accompanied by Chronic fibrous cholangitis with infiltration of abundant inflammatory cells dominated by mononuclear cells and eosinophils (Fig: 13). The accumulation of bile pigment in the bile canaliculi or dilated bile ducts was observed as yellowish brown concretions (Fig: 14). Portal triaditis (inflammation of hepatic triads and adjacent connective tissue) was also observed (Fig 15). Migratory tract with lymphocytic infiltration was observed in few sections (Fig: 16).

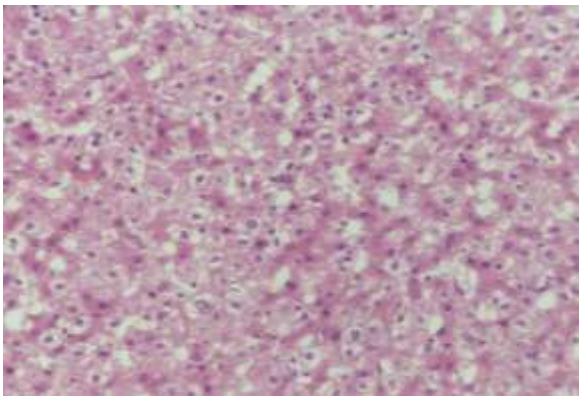

Fig. 6. Normal (uninfected) section of sheep showing normal hepatocytes (H and E stain).

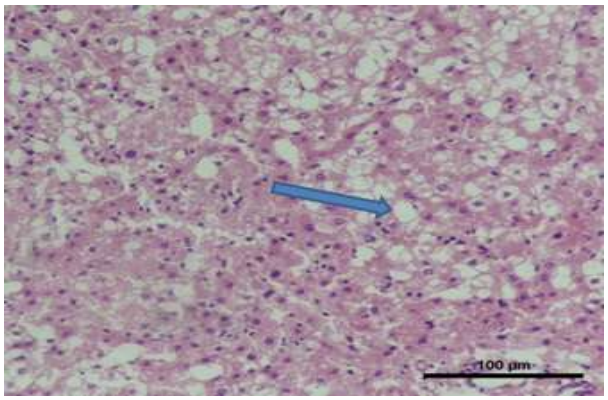

Fig 7. Fatty change with large clear vacuoles liver (H and E stain).

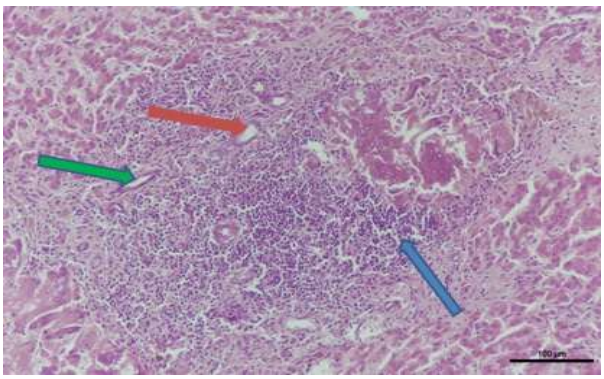

Fig 8. The chronic inflammation characterized by infiltrations of the mononuclear leukocytes (blue arrows), Atrophy (H and E stain).

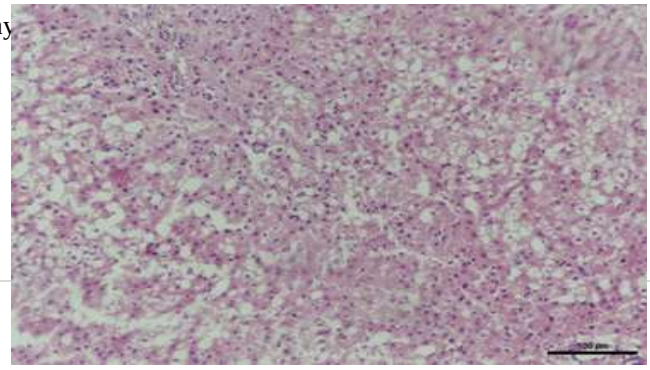

Fig 9. Acute/Parenchyal phase of fasciolosis showing vacuolar degeneration of hepatocytes with dilated hepatic sinusoids (H and E stain).

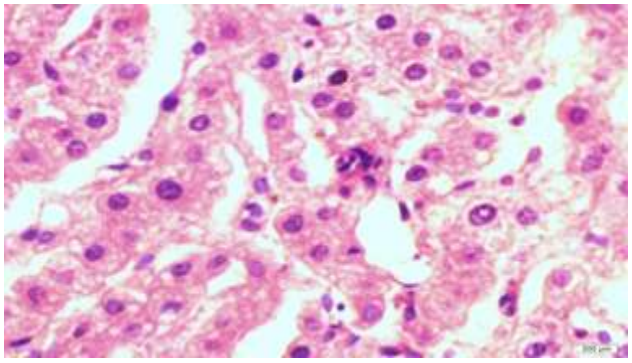

Fig 10. Liver section showing hepatocytes ballooning degeneration with periportal vein cellular infiltration, mainly eosinophiles, macrophages and lymphocyte (H and E stain).)

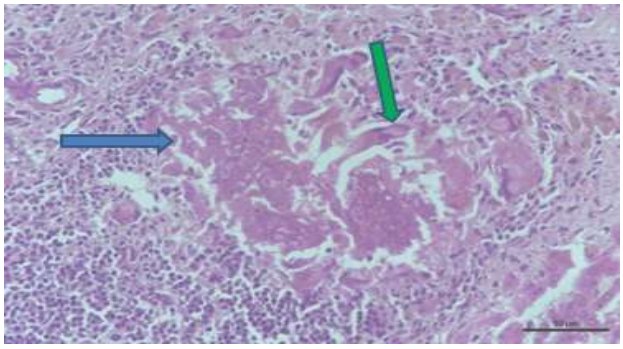

Fig 11. Portal fibrosis and compression atrophy of hepatocytes adjacent to fibrosis zone, accompanied by multinucleated giant cell formation (H and E stain).

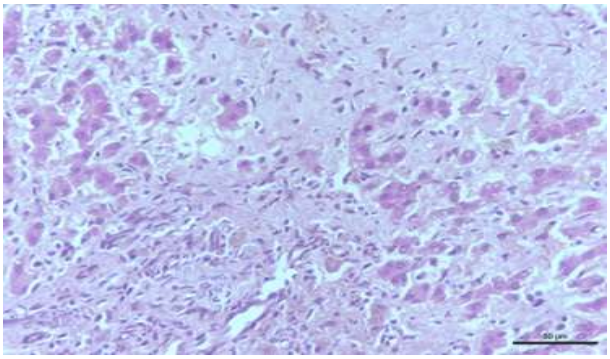

Fig 12. Individualization and distortion of the hepatic cells (H and E stain).

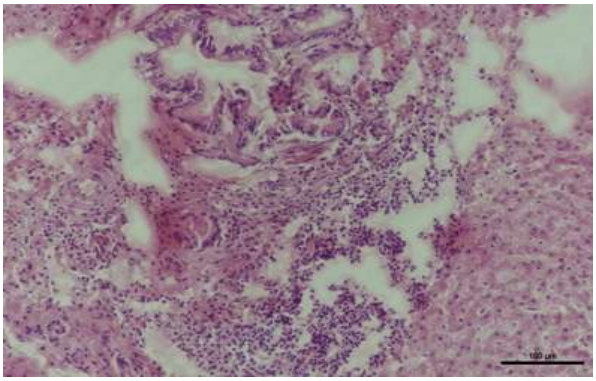

Fig 13. Bile duct hyperplasia and chronic fibrous cholangitis with abundant inflammatory infiltrate dominated by mononuclear cells and eosinophils(H and E stain).

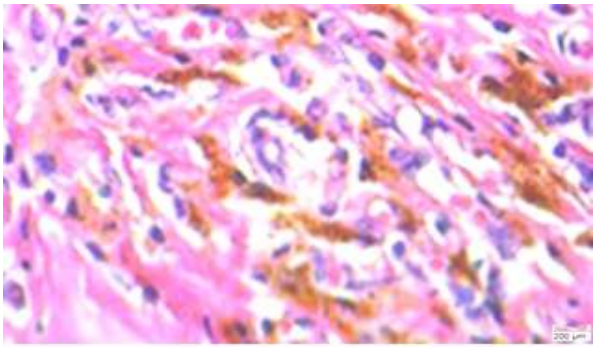

Fig 14. Yellowish brown materials accumulated among hepatocytes in bile canaliculi (H and E stain).

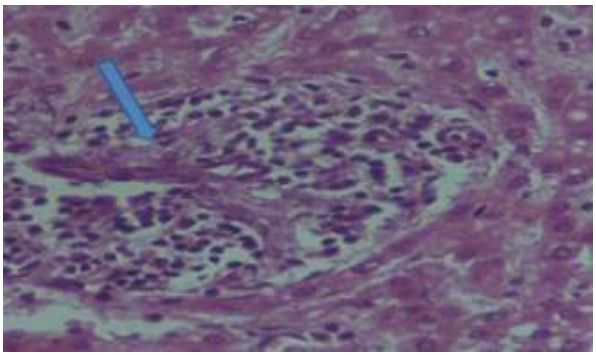

Fig 15. Portal hepatic triaditis (H and E stain)

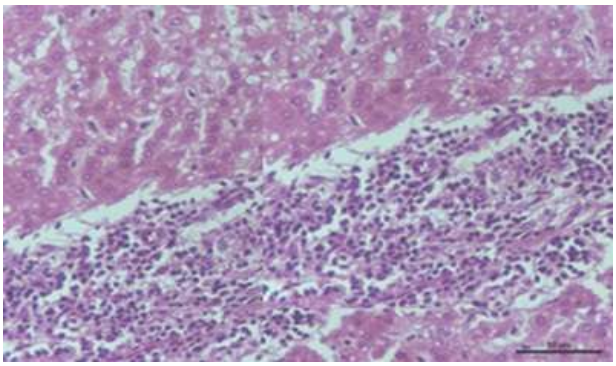

Fig 16. Migratory tract with lymphocytic infiltration (H and E stain)

Discussion

Liver is considered the most important organ for animal health production and reproduction. It is therefore important to evaluate the state of health of liver since this organ is involved in many disease processes either primarily or secondarily and also because any liver damage disturb metabolic processes that are vital for normal health and optimum productivity [17]. In the fasciolosis infected animals, the damage to the host species is either due to the mechanical or chemical effects by these parasites or by the hosts inflammatory and immune responses. The mechanical injury usually occurs when the infective metacercariae migrate through the liver capsule and hepatic tissue. The chemical effects involve a strong Th2 response, which in turn is associated with the production of cytokines thereby contributing to the overall pathophysiological condition. In the present study, we tried to find out the gross pathological and histopathological changes that occur in the sheep liver due to fasciolosis infection. Liver gross pathology in case of fasciolosis showed increase in the size of the organ (hepatomegaly) due to inflammatory changes in the parenchyma and hemorrhages on the parietal surface of the liver. These findings are in agreement with the report of [18], as they reported hepatomegaly and hemorrhages on the surface of the liver in chronic fasciolosis from dairy farm. The hemorrhagic foci on the surface represented the points of entrance of the immature parasites into the liver structure [19]. The hemorrhages may also occur due to inflammatory changes and migration of young flukes through liver parenchyma. Further we found paleness in some areas of liver which was due to the necrotic or damaged region. In some cases, there was congestion and firm whitish areas within parenchyma regarded as fibrosis. These results are partially identical to the findings of [20] who recorded pale and anaemic discolouration along with enlargement and necrotic foci at some affected parts of the liver. Similar results were also reported by [21-23]. We also recorded the gross pathological changes in bile duct which showed engorgement of bile in the duct. By cut section, the swollen and the fibrotic bile ducts were clearly visible and blocked by twisted flukes. The bile ducts were found filled with blackish brown exudate. It may be because of the presence of adult flukes in the bile duct which cause continuous irritation resulting hyperplastic proliferations and extensive ductular fibrosis.

Attributing to our finding [23], reported extensive enlargement and huge ductular fibrosis resulting the thickening of the bile ducts which also confirms the findings of [24]. These results were also in compliance with those mentioned by [25,26].

The histopathological changes in sheep liver by fasciolosis were characterized by infiltration of eosinophils, fibroblasts and lymphocytes in the area previously migrated by young flukes. The infiltration of these phagocytic cells occurs in case of chronic inflammation due to the release of proteases by the flukes which cause continuous irritation and migration through the liver parenchyma which leads to tissue damage by hemorrhages. Our results are in agreement with two previous studies of [27-32] who proved that the migration of immature liver flukes through the tissue causing hemorrhage and irritation, and brought the cellular inflammatory. Our results also showed the atrophy, necrosis and fatty changes in the liver sections due to chronic Fasciolosis, which were in consistent with [33] who reported atrophy, necrosis and fatty changes in chronic Fascioliasis. The atrophy and necroses of the tissue may be because of the digestion of the host components by the flukes though the release of proteases, which facilitates their migration, feeding as well as the immune evasion. We observed the vacuolar degeneration of hepatocytes along the migratory route of the parasite characterized by cell wall degeneration, deformed nuclei and cytoplasmic contents emptied in the sinusoids. Such mechanical hepatic damage was in partial agreement with the studies of [34] who observed parenchymal and hepatic tissue destruction accompanied by haemorrhagic lesions. Some sections showed hepatocytic degeneration with periportal vein cellular infiltration of mainly eosinophiles, macrophages and lymphocytes which was in full agreement with the studies of [30,32]. Infiltration of oesinophils coupled with the accumulation of macrophages and lymphocytes were the prominent features in this investigation particularly during the migratory phase of infection. Eosinophilia, characterized by increase in the number of eosinophils has been recognized as a distinguishing feature of helminth infections in mammals and has been used as an important diagnostic procedure for the parasitic infections. We also observed Portal fibrosis in the portal area which was characterized by extensive proliferation of fibrous connective tissue with infiltration of mononuclear inflammatory cells**.** In some cases it was accompanied by multinucleated giant cell formation formed by the fusion of macrophages that has the ability to engulf very large particles at the inflammation site. Similar results were also observed by [29,31,32] in case of chronic fasciolosis in bovine livers except that the multinucleated giant cell formation was not reported in their case. However, [30] reported the formation of multinucleated giant cell from histopathological studies on small ruminants.

Histopathological studies of bile duct showed the accumulation of bile pigment as yellowish brown concretions in the dilated bile ducts. Thickening of the bile ducts and fibrosis in a portal area due to chronic fasciolosis was also found. These might be because of the immunological reaction of macrophages and lymphocytes infiltration that merges with fibrotic healing of the necrotic areas during the later stage of fasciolosis. These results were similar to those obtained by [35,36,17,26]. Migratory tract with lymphocytic infiltration was more common

case. These results were in compliance with [37,23,19,31] who also found hemorrhagic migrating tracts with leucocytic infiltration mainly macrophages, lymphocytes and plasma cells in the hepatic parenchyma. Our results also revealed the hyperplasia of the bile ducts accompanied by chronic fibrous cholangitis with infiltration of abundant inflammatory cells dominated by mononuclear cells and eosinophils. These results agreement with the studies of obtained by [29-32]. Portal triaditis (inflammation of hepatic triads and adjacent connective tissue) was also observed in our case which was not reported by other authors in case of fasciolosis. In our study no multifocal nodules were noted and even less extensive ductular thickness was recorded in chronic form of fasciolosis. However, [30] reported focal and or multi focal liver nodules, from small ruminants which might be due to abnormal host immune response that resulted against Fasciola eggs, dead larvae or the fluke byproducts trapped into the liver parenchyma.

The differences in the types of histopathological lesions in fasciolosis by different workers can be due to different hosts, number and types of species present in the hosts, strength and type of immune response by the host and obviously the different environmental factors.

Conclusion

It could be concluded that the gross pathological and histopathological changes in the livers of sheep infected with fasciolosis reflects the extent of tissue damage which can predict the health status of the animal. Understanding the type of the damage in case of acute and chronic fasciolosis will help in finding ways for the preventive and therapeutic measures. Serious care and attention are required of both the veterinary workers and the public health planners to ensure that seriously damaged livers are not passed on for human consumption because of their deranged nutritional values and health risk problems. The grazing of cattle should be restricted to lesser snail infected sites to reduce the rate of animal infection and the consequent economic losses.

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