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Pathological studies on spontaneously occurring liver conditions of goats (*Capra hircus*) in southern region of Rajasthan

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Abstract
The liver is one of the main metabolic organs and, as such, is positioned between the digestive tract and the systemic circulation. Liver is affected by systemic disease conditions and bacterial pathogens. Also, some of the diseases liken hydatidosis of the liver have public health importance. As the liver is an edible organ that is highly priced, partial or total condemnation of liver leads to financial losses. of 14.98% (n=37), followed by Congestion 12,96% (n= 32), Chronic hepatitis 11.34% (n=28), Hepatic Abscess 8.91% (n=22), Cysticercosis 6.07% (n=15), Acute Hepatitis 5.67% (n=14), Cloudy Swelling 4.86% (n=12), haemorrhage 4.45% (n= 11), Fatty Changes 4.05% (n=10), Hydatid cyst 3.64% (n=9), Hydropic degeneration 2.02 (n=5), Telangiectasis 2.02 (n=5), Bile duct hyperplasia 2.02 (n=5), Hemosiderosis 0.81% (n=2), Amyloidosis 0.81% (n=2). The incidence of parasitic conditions recorded was 24.70% (61 cases).

Keywords: goat, incidence, liver, condemnation

1. Introduction
Goats (*Capra hircus*) have played a very vital role for man since prehistoric times. They were probably among first animals to be domesticated around 9000- 7000 BC. Goats are one of the dependable sources of income and provide nutritional support to the people living in the arid, semi-arid, hilly and tribal areas. In the country's goats make a very valuable contribution, especially to the poor in the rural areas. It is believed that goats are intelligent, independent, agile and tolerant to many diseases and parasites and can look after themselves much better than other livestock species.
Liver function is only impaired once more than 80% of the liver has been damaged. However, the liver does possess a unique capacity for maintaining its specific functions and simultaneously repairing and regenerating its own tissue. Liver is the first organ of the body to undergo pathological changes whenever an animal suffers from any disease and is the last organ to recover normally. As any other organ liver, could suffer from damages due to viral, bacterial, parasitic and fungal agents. In addition, several chemicals, drugs, environmental pollutants including insecticides, pesticides, fungicides and herbicides could cause liver damage when inhaled, ingested, injected or absorbed through skin.

2. Material and Methods
2.1 Sample collection: During the period from November 2018 to October 2019, A total of 1488 goats' liver of either sex, breeds and of different age groups were screened. On the presence of gross pathological abnormalities, morphology in terms of shape, size, colour, consistency, odour, location and type of lesion in individual part of the liver, as far as possible the colour of tissue was noted immediately after collection and prior to fixation. A total of 247 animals showed various gross lesions of pathological significance in the liver that formed the source of material for the present study. The samples were collected from carcasses of goats subjected to post-mortem examination to various veterinary clinics and various slaughter houses located in Udaipur, Dungarpur, Chittorgarh and Rajsamand districts of southern Rajasthan.

2.2 Histopathology: Formalin-fixed tissues were processed routinely, dehydrated through graded ethanol (70, 80, 90, 95 and 100%), cleared by xylene and impregnated and embedded

in paraffin wax. Sections were cut at 4-5 µm (micron) thickness with the help of semi-automatic rotary microtome (Lilliie, 1965) [14]. The sections were stained with haematoxylin and eosin (H&E) stain following conventional procedure (Luna, 1968) [13].

2.3 Parasites identification: At the time of slaughter/ necropsy, liver was examined for presence of parasites were collected in 10% normal saline and were processed as per the standard procedures (Soulsby, 2012) [23] for morphological identification.

3. Results and Discussion

In the present study, a total of 1488 goats of irrespective sex, different age groups and breeds in southern region of Rajasthan including district Udaipur, Dungarpur, Chittorgarh, and Rajsamand. On gross and histopathological examination, definitive lesions of various types were found in 247 (16.60%) livers out of 1488 examined (Table 1.).

The present study revealed congestion was encountered in 32 cases with an occurrence of 12.96%. Grossly, liver was congested over the visceral lobe side, appearing bright red in colour, with swollen lymph node and distended gallbladder. On cut section showed congested red centres surrounded by pale coloured unaffected peripheral areas. Microscopically, liver showed severe congestion in central vein and nearby sinosoids and the sinusoid filled with RBCs. In some cases, there were chronic venous congestion, the central vein was enlarged and round. Haemorrhages were encountered in 11 cases with an occurrence of 4.45%. Grossly, the affected liver showed petechial or ecchymotic hemorrhage on the surface with distended gallbladder. Microscopically, liver showed haemorrhage, extravassation of RBCs in the hepatic parenchyma along with infiltration. A zone of subcapsular haemorrhage was also noticed. In few cases with haemorrhages focal area of necrosis with loss of cellular details and architecture and sever neutrophilic infiltration and were observed. Some places showed dilated engorged blood vessels and sinusoids, sever diffuse necrobiotic changes in hepatocytes with peri vascular mononuclear inflammatory cell infiltration. The hemosiderosis was encountered in 2 case with an occurrence of 0.81%. Grossly, no appreciable gross lesions were observed except presence of pale to reddish brown coloured areas on liver surface. Microscopically, liver showed hemosiderosis, brown coloured hemosiderin scattered in liver parenchyma. In some places severe haemorrhages were noticed, in centre of which hemosiderin accumulated. Etiology for hemosiderosis may be excessive amount of iron in feed or have iron compounds ingested for the prevention of anaemia. Cloudy swelling was encountered in 12 cases with an occurrence of 4.86%. Grossly, the liver showed cloudy swelling, slightly Pale, friable and enlarged with rounded border. The cut surface bulged out slightly on incision and the cut edges of the capsule retracted. Microscopically, liver showed cloudy swelling, hepatocytes are swollen with fine granulation in the cytoplasm and reduce sinusoidal space. In some cases, cells were swollen and their edges was rounded, cytoplasm stained slightly more intense with eosin. The internal structures of the cells were slightly hazy. Hydropic degeneration was encountered in 5 cases with an occurrence of 2.02%. Grossly, the affected liver showed dark colour with a pronounced zonal or lobular pattern, caused by periacinar or centrobular hydropic degeneration of hepatocytes (Fig. 1). Microscopically, hepatocytes with presence of hepatocytes with presence of small clear, multiple vacuoules. In some cases, near this area cloudy swelling and necrotic changes were also observed. Fatty changes were encountered in 10 cases with an occurrence of 4.05%. Grossly, the affected liver showed enlarged friable, yellow colour due to excess fat with round border. The capsules were tensed with indistinct edges and prominent grooves of lobes were present. Consistency of the liver was soft and doughy. On sectioning with sharp knife, the cut surface usually bulges with greasy texture to touch and droplets of fat are visible on the blade of knife. Microscopically, well defined, presence of clear fat vacuoles in the cytoplasm of hepatocytes and few hepatocytes seen as signet ring appearance. Fatty changes were mainly seen around central vein as well as around the portal triad. The sinusoidal space was narrowed and, in few places, there were necrotic changes in the hepatocyte and some hepatocytes were atrophied. Liver necrosis was encountered in 37 cases with an incidence of 14.98%. Grossly, the affected liver showed haemorrhages and necrotic patches dark red in colour, distended gall bladder with bile juice (Fig. 2). At places, pale patches of necrotic area were also visible. The colour of the liver was either pale or mottled and the surface was rough with sharp edges. Microscopically, liver showed areas of focal necrosis with small patches surrounding by cellular infiltration (Fig. 3). Liver amyloidosis was encountered in 2 case with an occurrence of 0.81%. Grossly, the liver affected was enlarged, round edges, has doughy consistency, pits on pressure, and has a cyanotic yellow colour. In some cases, the liver very friable, rupture easily, haemorrhage was observed. Microscopically, liver showed amyloidosis, amyloid deposited around dilated central vein and give homogenous pink staining, glassy appearance (Fig. 4). In hepatocytes severe deposition of amyloid around central vein and in sinusoidal space causing pressure atrophy on nearby hepatocytes and give homogenous pink staining, glassy appearance.

In the present study, the inflammatory conditions comprised of 65 (26.32%) cases out of 247 liver lesions examined. Acute hepatitis was encountered in 15 case with an occurrence of 6.07%. Grossly, the affected liver was enlarged, congested and pale areas on the surface. In some cases, livers were found soft and fragile also. Microscopically, lesions were characterized by congestion, scattered haemorrhages and infiltration of large number of neutrophils and few lymphocytes around the hepatocytes that have undergone necrosis. In hepatocytes seen dilation of central vein, infiltration of polymorphonuclear cells around central vein and focal necrosis. Besides these, hepatocytes were swollen with granular cytoplasm indicative of degeneration and necrosis. The surviving hepatocytes were smaller, elongated and have lost their normal architectural details at places. Chronic hepatitis was encountered in 28 case with an occurrence of 11.34%. Grossly, the affected liver showed perihepatitis (Glissonian's cirrhosis) dark in colour and thickened Glisson's Capsule (Fig. 5). In some cases, enlarged with a mottled and bile-stained surface, fibrosis and nodules formation. Microscopically, lesions in chronic hepatitis in general and cirrhosis in particular were characterized by increase in fibrous connective tissues within and around the lobules and conversion of normal liver architecture into structurally abnormal lobules. In chronic hepatitis, thickened Glisson's capsule with fibrosis extend from the capsule to a distance beneath of liver parenchyma (Fig. 6). In the hepatocytes, fibrous tissue invades the parenchyma and

encircles the individual hepatocytes (Fig. 7). Hepatic abscess was encountered in 22 case with an occurrence of 8.91%. Grossly, the affected liver showed focal multiple whitish abscess with single to multiple in numbers and 1.9 cm in diameter (Fig. 8) and also embedded in the liver parenchyma. It contains thick and creamy dry, inspissated as well as semisolid pus. Microscopically, liver with abscess showed thick central cellular necrotic debris surrounded by cellular infiltration and surround by finely granular eosinophilic and basophilic substances infiltrated with neutrophils (Fig. 9). In some cases, loss of hepatocytes with cellular infiltration including plasma cells, neutrophils, leucocytes. Central 'necrotic area contained degenerated neutrophils and cellular debris. There were extensive degenerative and necrotic lesions observed in hepatic parenchyma.

In the present study, the parasitic conditions comprised of 61 (24.70%) cases out of 247 liver lesions examined. Fasciolosis was encountered in 37 case with an occurrence of 14.98%. Grossly, the affected liver showed fasciolosis and Fasciola parasite in the bile duct (Fig 10). On cut section of liver showed hard indurated bile duct forming tract like structure containing parasite in side (Pipe stem liver). The flukes recovered from all the cases were morphological identified as *Fasciola hepatica*. In fasciolosis also gross lesions noticed in the liver were enlarged, friable, icteric with areas of necrosis, haemorrhages and mottling that showed few haemorrhagic tracts under the capsule. The livers were enlarged, friable and revealed dark areas of necrosis, haemorrhages and haemorrhagic tracts of migration of flukes grossly observed. Microscopically, liver with fasciolosis showed cross section of liver fluke within the liver parenchyma (Fig. 11). The parasites present in bile duct along with hyperplastic bile duct and fibrino necrotic layer. The degenerative bile duct hyperplasia with fibrino necrotic layer. In a few cases, severe vascular and sinusoidal congestion was noticed. Bile ducts revealed desquamation of epithelium and infiltration of eosinophils, macrophages and lymphocytes within the wall of bile duct and also in the portal tracts. The flukes recovered from all the cases were morphologically identified as *Fasciola hepatica*. Cysticercosis was encountered in 15 case with an occurrence of 6.07%. Grossly, the affected liver showed Cysticercus cyst adhered to the liver outer surface a distended Gall bladder with greenish bile juice (Fig. 12). The cysts were fluid filled and contained a small, solid, whitish scolex inside the cyst that was visible through the translucent wall. Microscopically, at the site of attachment, the hepatic parenchyma revealed infiltration around portal vein (Fig. 13). In some cases, a few focal areas of necrosis were observed. The overall hydatid cyst was encountered in 9 case with an occurrence of 3.64%. Grossly, the affected liver showed multiple small hydadtid cyst embedded in the liver parenchyma (Fig. 14). The livers revealed solitary (single) hydatid cysts of variable size in the middle of the liver. The consistency of liver was firm and cut surface did not bulge. Microscopically, hydatid cyst with clear watery fluid revealed large cystic spaces with a clear hyaline wall surrounded by fibrous connective tissue capsule and infiltration of lymphocytes. Due to the presence of cyst, the adjoining parenchyma showed pressure atrophy. The hydatid cyst wall around zone of inflammatory reaction with leucocytes and surrounded by fibrosis (Fig. 15). Besides these, hepatic parenchyma exhibited varying degree of degenerative changes. In some cases, there was bile duct hyperplasia and regeneration of bile ducts.

In the present study, the miscellaneous conditions comprised of 10 (4.05%) cases out of 247 liver lesions examined. Telangiectasis was encountered in 5 case with an occurrence of 2.02%. Grossly, the affected part appeared liver appeared as dark red area, irregular in shape but well circumscribed and ranging from pinpoint to many centimetres in size. Microscopically, liver showed sinusoidal space widely dilated and with loss of hepatocytes. Bile duct hyperplasia was encountered in 5 case with an occurrence of 2.02%. Grossly, the affected livers were enlarged with presence of light and dark patches were clearly visible on the surface of the liver. On the cut section, the swollen and the fibrotic bile ducts were prominent. Microscopically, liver lesion severe destruction in the liver tissue including inflammation, atrophy, necrosis, fibrosis and hyperplasia of the bile ducts. The cellular infiltration of the inflammatory cells including macrophages and plasma cells were observed in section. The infiltration of these cells was severe in the area of portal triads. Bile ducts manifested hyperplastic changes in the epithelium. The bile duct hyperplasia, dilated central vein, congestion, fibrosis and proliferation of numerous small to large size the bile ducts (Fig. 16)

Table 1: Overall occurrence of various spontaneous liver lesions in goats (*Capra hircus*) at Udaipur district of Rajasthan

Sr. No	Pathological Conditions	No. of conditions	Percentage (N= 247)
1	Circulatory Disturbances	45	18.22
1.1.	Congestion	32	12.96
1.2.	Haemorrhages	11	4.45
1.3.	Hemosiderosis	2	0.81
2	Degenerative changes	66	26.72
2.1.	Cloudy Swelling	12	4.86
2.2.	Hydropic Degeneration	5	2.02
2.3.	Fatty change	10	4.05
2.4.	Necrosis	37	14.98
2.5.	Amyloidosis	2	0.81
3	Inflammatory Conditions	65	26.32
3.1.	Acute Hepatitis	15	6.07
3.2.	Chronic Hepatitis	28	11.34
3.3.	Hepatic Abscess	22	8.91
4	Parasitic Conditions	61	24.70
4.1.	Fasciolosis	37	14.98
4.2	Cysticercosis	15	6.07
4.3.	Hydatidosis	9	3.64
5	Miscellaneous conditions	10	4.05
5.1.	Telangiectasis	5	2.02
5.2.	Bile duct Hyperplasia	5	2.02
Total		247	

Fig 1: Gross Photograph of liver showing dark colour with a pronounced zonal or lobular pattern, caused by periacinar or centrobular hydropic degeneration of hepatocytes

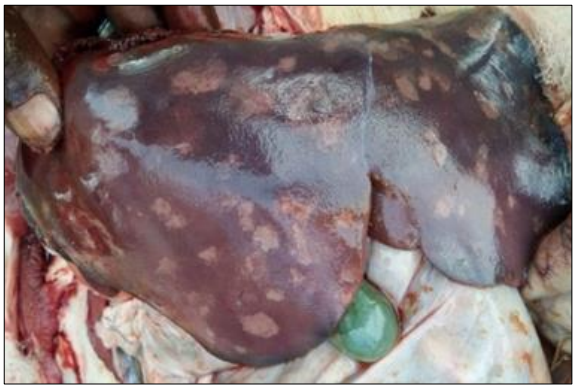

Fig 2: Gross Photograph of liver showing haemorrhages and necrotic patches dark red in colour, distended gall bladder

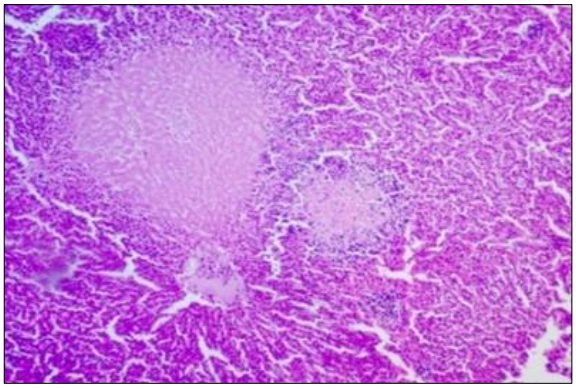

Fig 3: Microphotograph of liver showing areas of focal necrosis with small patches surrounded by cellular infiltration. H&E- 100X

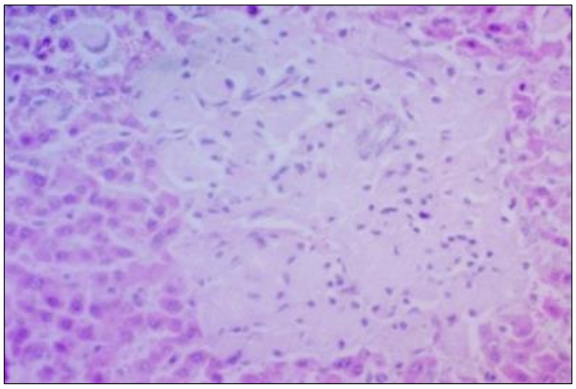

Fig 4: Microphotograph of liver showing amyloidosis, amyloid deposited in central vein, glassy appearance. H&E- 400X

Fig 5: Gross Photograph of liver showing perihepatitis (*Glissonian cirrhosis*) dark in colour and thickened Glisson's Capsule.

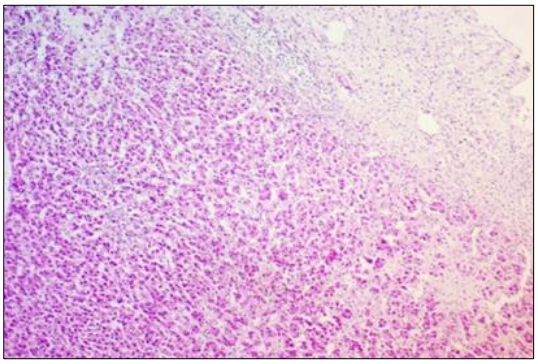

Fig 6: Microphotograph of liver showing chronic hepatitis, thickened Glisson's capsule with fibrosis extend from the capsule to a distance beneath of liver parenchyma. H&E- 100X

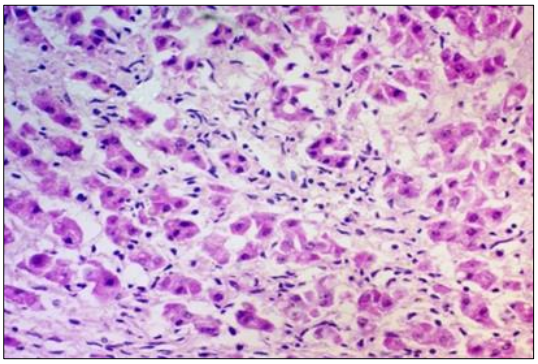

Fig 7: Microphotograph of liver showing pericellular cirrhosis with monocytic infiltration and fibrosis. Fibrous tissue invades the parenchyma and encircles the individual hepatocytes. H&E- 400X

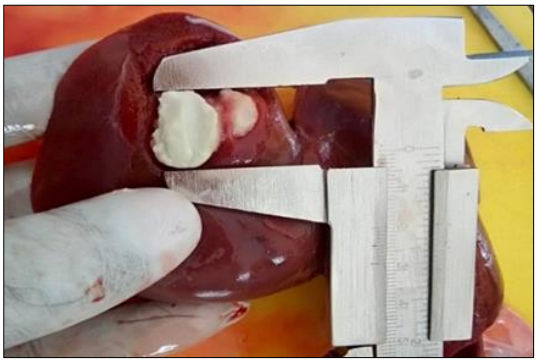

Fig 8: Gross Photograph of liver showing hepatic abscess 1.9 cm in diameter

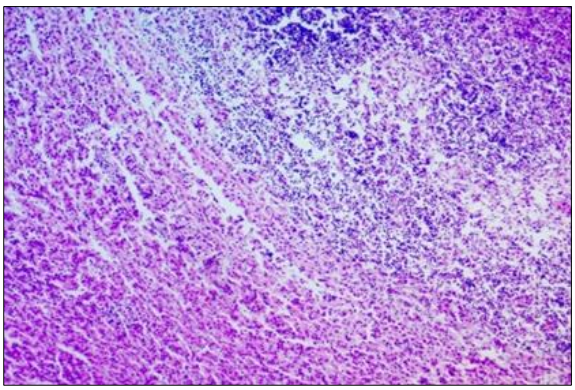

Fig 9: Microphotograph of liver showing abscess surrounded by finely granular eosinophilic and basophilic substance infiltrated with neutrophils. H&E- 100X

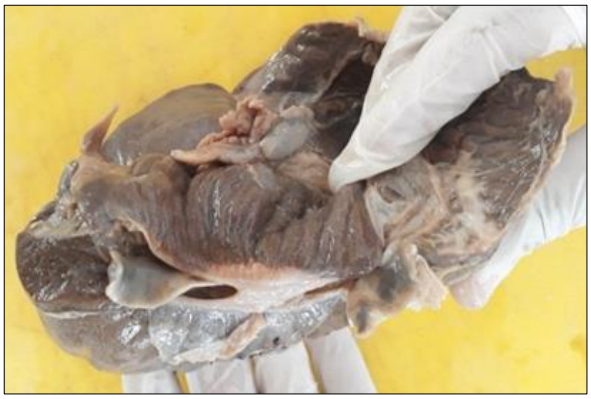

Fig 10: Gross photograph of liver showing fasciolosis, Fasciola parasite in the bile duct

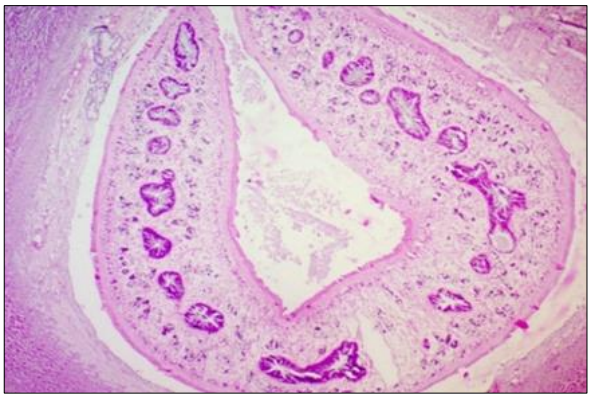

Fig 11: Microphotograph of liver showing cross section of liver fluke within the liver parenchyma. H&E- 40X

Fig 12: Gross Photograph of liver showing *Cysticercus cyst* adhered to the liver outer surface and distended Gall bladder

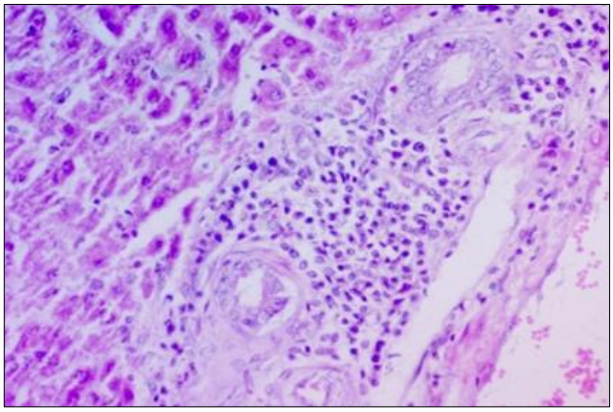

Fig 13: Microphotograph of liver section infected with cysticercosis showing infiltration around the portal vein. H&E- 400X

Fig 14: Gross photograph of liver Hydatidosis showing multiple small hydadtid cyst embedded in the liver parenchyma

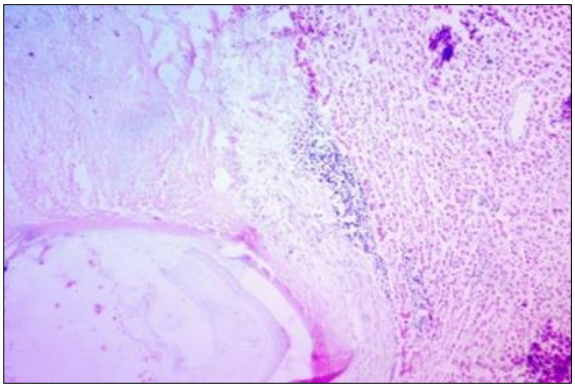

Fig 15: Microphotograph of liver showing hydatid cyst, hydatid wall around zone of inflammatory reaction with leucocytes and surrounded by fibrosis. H&E-100X

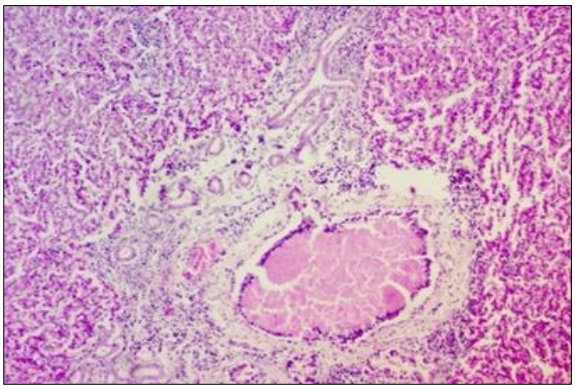

Fig 16: Microphotograph of liver showing bile duct hyperplasia, dilated central vein with congestion, fibrosis and proliferation of numerous small to large size the bile ducts. H&E- 100X

4. Discussion

In the present study, a total of 1488 goats examined based on gross and histopathological examination hepatic lesions of pathological significance were recorded in 247 goats with an overall occurrence of 16.60%. Which was in consonance with the observation made by In India, Ravi Kumar *et al.* (2006) [18], Sanjeeth *et al.* (2015) [20]. In abroad, Mellau *et al.* (2010) [15] and Gezu *et al.* (2014) [6] reported. This variation in the occurrence might be due to the differences in the age, sex, breed of goats, geographical area and environmental factors of the area under study. The study revealed that Necrosis and Fasciolosis was the most common condition with an incidence of 14.98% followed by Congestion 12,96%, Chronic hepatitis 11.34%, Hepatic Abscess 8.91%, Cysticercosis 6.07%, Acute Hepatitis 5.67%, Cloudy Swelling 4.86%, haemorrhage

4.45%, Fatty Changes 4.05%, Hydatid cyst 3.64%, Hydropic degeneration 2.02, Telangiectasis 2.02, Bile duct hyperplasia 2.02, Hemosiderosis 0.81%, Amyloidosis 0.81%.

The parasitic conditions were noticed in 24.70% of liver that comprised of Fasciolosis 14.98% (*Fasciola hepatica*) Cysticercosis 6.07% and Hydatidosis 3.64%. Fasciolosis was recorded in 37 case with an occurrence of 14.98%. The similar finding reported by Mellau *et al.* (2010) [15] and Amit Khajuria *et al.* (2013) [12]. In abroad, Hassan *et al.* (2014) [9] and Belina *et al.* (2015) [3] reported. The gross and microscopic picture of fasciolosis observed in the present study was in agreement with the findings by Belina *et al.* (2015) [3], Dharnesha *et al.* (2015) [4], Rakesh Kumar *et al.* (2015). The Lesion of fasciolosis in goats due to *Fasciola hepatica* were described earlier in India by Sanjeeth *et al.* (2015) [20] and Ghosh *et al.* (2016) [7]. The severity of pathological changes probably depends on the number of fluke's present, the host species and breed (Howlader and Huq, 1997) [10]. Progressive irritation by adult fluke wondering in the biliary tract cause biliary inflammation like hyperplasia, duct wall thickening and dilatation. Mechanical irritation of hepatocytes by the fluke in chronic fasciolosis enhances hepatocyte destruction with persistent healing that result in fibrous connective tissue proliferation leading to hepatic cirrhosis. The obstruction to intrahepatic bile flow leads to extreme bile ductular proliferation, inflammation and necrosis of adjacent parenchyma, portal scarring and bridging fibrosis. Eosinophilia occurs due to sensitivity due to foreign protein of parasite which might be a part of immune phenomenon (Belina *et al.*, 2015) [3]. Cysticercosis was recorded in 15 case with an occurrence of 6.07%. In India, cysticercosis in goat liver was reported previously by various authors with an occurrence by Amit Khajuria *et al.*, (2013) [12], Sanjeeth *et al.*, (2015) [20] and Singh *et al.*, (2015) [21]. In abroad, the occurrence reported by Jibat *et al.*, (2008) [11] and Mellau *et al.*, (2010) [15]. Singh *et al.* (2015) [21] opined that the difference in the occurrence might be due to the difference in managemental, environmental and other factors. Etiologically, it is a cystic stage of *Taenia hydatigenia* which is found in the small intestines of dogs and cats and the ova passed with faeces are ingested by intermediate hosts such as sheep and goat with pasture contaminated with eggs. After ingestion, the larvae penetrate the small intestine and disseminate to various tissues especially liver, omentum, mesentry and peritoneum and on the liver surface they develop into thin walled fluid filled bladders. The developing cysticerci migrate in the liver causing haemorrhagic and fibrotic tract. Mature cysticercus in the peritoneal cavity usually causes no harm (Bejiga *et al.*, 2016) [2]. Hydatidosis was recorded in 9 case with an occurrence of 3.64%. In India, hydatidosis was reported by Sangaran *et al.* (2014) [19], Moudgil *et al.* (2018) [16], Godara *et al.* (2014) [8], Sanjeeth *et al.* (2015) [20] reported. In abroad, Osman *et al.* (2014) [17], Bayu *et al.* (2013) [1] and Faraji *et al.* (2015) [5] reported.

In the present study, miscellaneous conditions were noticed in 4.05% of liver that comprised of Telangiectasis 2.02% and Bile duct Hyperplasia 2.02%. Telangiectasis was recorded in 5 case with an occurrence of 2.02%. The occurrence of telangiectasis observed in the present study was almost similar to that (2.3%) reported by Atasever *et al.* (2000). Bile duct hyperplasia was recorded in 5 case with an occurrence of 2.02%. The recorded observations are similar to those described by Rakesh Kumar *et al.* (2015), Sonawane *et al.* (2016) [22] and Moudgil *et al.* (2018) [16].

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