Studies on

# THE INCIDENCE OF PATHOLOGICAL CONDITIONS IN CAPRINE LIVER

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*Thesis*

*Submitted to*

**Magadh University**

In Partial Fulfilment Of The Requirement For The Degree Of

M. Sc. ( Vet. )

*IN*

**PATHOLOGY**

*By*

*Purendra Narayan Singh*

B. V. Sc. & A. H.

POST GRADUATE DEPARTMENT OF PATHOLOGY

BIHAR VETERINARY COLLEGE,

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# STUDIES ON THE INCIDENCE OF PATHOLOGICAL CONDITIONS IN CAPRINE LIVER.

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P A T H O L O G Y

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December , 1967 .

Prof.P.B.Kuppuswamy
B.A.,G.M.V.C.,B.V.Sc.,
P.G.(Newzealand),M.S.(Missouri),
Principal and Head of the Department.

Post Graduate Deptt.of Pathology
Bihar Veterinary College,
PATNA.

This is to certify that the entire work presented in Thesis entitled " Studies on the incidence of Pathological conditions in Caprine Liver " is the bonafied work of Sri Purendra Narayan Singh, a candidate for the degree of M.Sc.(Vet) with Pathology as his major subject, which was carried out under my supervision and guidance.

( P.B. Kuppuswamy )

23/12/69

# A C K N O W L E D G E M E N T S.

The author expresses his deep sense of gratitude and thankfullness to Sri P.B.Kuppuswamy,B.A., G.M.V.C., B.V.Sc., P.G.(Newzealand), M.S.(Missouri,U.S.A.), Principal and Head of the Department of Pathology and Bacteriology for his valuable guidance, constant supervision, encouragement and advice throughout the work.

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( P.N.S.)

# C O N T E N T S.

# I N T R O D U C T I O N

The liver is considered to be the largest gland performing a great number and varieties of functions. In structure it is one of the simplest and in function one of the most complex organ in the body. More than ninety years ago Rokitansky in his pathological anatomy remarked that " the diseases of the liver have continued to remain to the present day a subject of extreme difficulty". ( Boyd 1947).

The organ is intimately concerned with many physiological functions.

It will not be out of place to mention that liver is an important nutritious diet for human being and that is why it is largely used for human consumption specially goat liver. As liver contains anti permicious anaemia factor it is largely used for manufacture of medicines.

The organ recieves blood supply from arterial and veinous sides.The anatomical association of the different structures of the liver lobule ; such as hepatic cells, blood vessels and bile passages are such that onset of disturbance or any pathological condition in any one structure sooner or later spreads to all parts of the lobule resulting the involvment of the entire organ or large part of it.

Moreover liver is the seat of predilection for many helminth parasites such as species of Fasciola, Dicrocoelium, Amphistomes etc. The larvae of various nematodes also migrate through the liver causing damage to the liver tissue. Moreover

liver recieves all the toxins for the purpose of detoxification. Liver therefore is alsways prone to various injuries and any damage to it bars it from performing its normal functions.

So it is surprising that literature on pathological conditions of liver among the domestic animals particularly in goats is quite scanty.

The goat has been appropriately termed " The poor man's cow " and certainly no better designation could be found to express the position of this useful creature amongst the domestic animals. It is very valuable for its manure, milk, fleece, guts hide and meat. Among the Indian goats some breeds like Jamunapari, Beetal and Barbari are well known for their milk character.The annual production of milk in India is 560 million maunds of which, goat milk comprises about 15 million maunds (Randhwa 1962) . On national level there are over 80 million goats in India yielding about 1,60,000 tons of meat annually ( Randhwa loc. cit) .In Bihar state there are over 7 million of goats . Goat meat is relished more than mutton. The value of the meat produced annually in this country has been estimated at 30.5 crores at the average rate of Rs. 70.00 per maund ( Lall - 1953).

Goat is very prolific. It gives birth to twins and even triplets twice in 14 months. Due to its rapid growth, quick maturity and high prolificity goats are considered to be one of the economical enterprises earning a good devident to the farmers and the goat breeders.

Presently India is facing acute problem of food scarcity owing to variety of causes viz. low production, increased population and natures' vagary in the form of drought and flood. We are therefore naturally more concerned with increased production in all spheres of Agriculture and Livestock industry. The problem of food scarcity can be solved by adopting scietific methods in Agriculture and livestock production.

Among all the livestock, goat occupies an unique position in livestock economics as it is one of the cheap sources of best quality milk required by young gernation and meat is one of our principal source of protein which is essentially required for the entainence of our nation's health and multiplies rapidly.

So in view of the importance of goat, liver of goat was selected for study of the detailed macroscopic and microscopical alterations encountered by it, so as to evolve suitable diagnostic, treatment and control measures against the same for the successful new development of goat husbandry.

## M A T E R I A L S and M E T H O D S :

In the present study the specimens of livers having gross lesions were collected from goats from different slaughter houses at Patna, from Livestock research station, Patna where the goats are slaughtered for manufacture of Rinderpest vaccine and from the autopsy cases performed in the department of Pathology ,Bihar Veterinary College, Patna.

Altogether 2,546 goats were examined and livers showing gross lesions were collected from 244 cases.The specimens were collected mostly from male goats.

Among the specimens collected, different degrees of gross lesions were encountered .As such, it was catagorized into different groups so that a selective incidence of each pathological lesion could be dealt with.

The different lesions thus encountered are tabulated below:-

....( Cont... )

Table No. 1.

| Serial No. | Pathological conditions encountered. | No. of animals from which specimens collected. | Percentage. |
|---|---|---|---|
| 1. | Biliary amphistomiasis | 1 | .039 |
| 2. | Fascioliasis | 82 | 3.22 |
| 3. | Hydatidiosis | 80 | 3.15 |
| 4. | Congestion | 71 | 2.78 |
| 5. | Icterus | 3 | .117 |
| 6. | Caseated and necrotic liver. | 3 | .078 |
| 7. | Abcess. | 1 | .039 |
| 8. | Eosinophilic hepatitis | 1 | .039 |
| 9. | Tubercle like nodule | 2 | .078 |
| Total. | | 244 | |

After each collection gross pathology of individual specimen was noted, marked with identifying numbers, gross photographs, where ever necessary were taken and livers were reserved in 10% formol saline solution in order to prevent putrifactive and autolytic changes. Formol saline which is an ideal fixative was used throughout the present study. It peneterates, kills, fixes and hardens the tissue quickly so that they can resist dehydration, cleaning staining and mounting during the process.

Small thin pieces of tissue measuring a few millimeter in thickness according to the lesions were cut and

preserved for another 24 hours to ensure perfect fixation.

Fixed tissues were washed thoroughly in running tap water for 24 hrs, dehydrated in ascending grades of alcohol, cleaned in several changes of Xylol and then transfered to paraffin having melting point 57°C. The temperature of the paraffin bath which was mentained for constant temperature at 60°C and three changes in melting paraffin were done for infilteration of paraffin into the tissues. Finally blocks were made by tissues embedding in paraffin.

Paraffin sections were cut 5 /u in thickness and the ribbon thus procured were shifted to water bath having a temperature of 45° C .The floating sections were collected on clean slides previously semeared with Mayer's glycerine eggalbumin.

Tissue sections were deparaffinized by passing it into three changes of Xylol and then brought to water level by passing it into descending grades of alcohol and finally into distilled water.

Tissue sections were fainally stained with Harri's alum haematoxylin and counter stained by 1% alcohilic eosin. For the identification and demonstration of special structures abnormally present or developed due to altered tissue reaction, the sections were stained with special stains viz:-

(i) Van Gieson's stain ( Culling , 1957) for collagen fibres;

(ii) Lievaditi's method ( Culling , 1957 ) for staining spirochaetes in blocks.

(iii) Gram's stain ( Culling 1957 ) for Gram positive and Gram negative bacteria.

( iv) Perl's Prussian blue reaction ( Culling 1957) for haemosiderin.

Finally all stained sections were dehydrated in ascending grades of alcohol, cleaned in three changes of Xylol and were mounted permanently in Canada balsam.

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# O B S E R V A T I O N S

## BILIARY AMPHISTOMIASIS

## I N T R O D U C T I O N.

Gigantocotyle explanatum(Creplin 1847) the only parasite of the family paramphistomidae is found commonly in liver of domestic ruminents in India. Creplin ( 1847) discovered this parasite from the bile duct and gall bladder of Bovs tauri. Fischoeder ( 1904) recorded this parasite from the bile ducts of the Zebu at Berlin and also Buffelus indicus of Saigon (Cochin-China) . Dawes ( 1936) examined some specimens of this parasite from the bile ducts of Malayans cattle, Buffalo and goats. Srivastav ( 1945) also recorded this parasite from both the hosts but he did not mention the location of this parasite in the host. Kulesiri and Seneviratne ( 1956) recorded this parasite from the bile duct of buffaloes in Cylon and also studied the gross lesions and hsto- pathology of the liver. Verma ( 1957) recorded this parasite from the bile duct of only buffaloes in Bihar, India. Singh ( 1958) recorded this parasite from the liver, bile duct, gall bladder of the domestic ruminents in India but no mention is made of goats.

Available literature shows that no one has recorded this parasite from goat in India. But during the present investigation one case harbouring this parasite and causing bliary amphistomiasis in goat was recorded.

## REVIEW OF LITERATURE.

No systematic work on the various changes encountered in the liver of goats affected with Amphistomes appears to have been done. Kulasri and Seneviratne ( 1956) had studied the gross lesions and histo pathology of liver of buffaloes affected with amphistomes . Gross and histological lesions in the liv r of goats infected with Gigantocotyle explanatum were studied in the present work.

## R E S U L T S

### Gross Pathology:-

The capsule was thicknened and opaque. There were few haemorrhagic spots on the surface ( Figxx.1 ) The common bile duct was very much distended and numerous amphistomes were seen in its lumen. Some of them were lying free in the lumen but most of them were attached to the mucosa of the bile duct.The wall of the bile duct was thickened and on separation of the amphistome from the mucosa, a large numer of nodule like structures each of the size of the lentile became apparent (Fig. 2) . Kulasri and Senevdratne ( 1956) did not find any parasite in the gall bladder of buffaloes but in the present studies one parasite was found lying free in the gall bladder.The gall bladder was distendéd with thick turbid and blood tinged bile.The wall of the g ll bladder was slightly thickened.

Histopathology:-

Sections of liver showed increased capsular thickening composed of alaminated collagenous memberane and fibroblastic layer. Immediatly under neath strands of fibroblasts stretching deep into the liver substance were seen. Specimen revealed presence of haemorrhagic tracks . The tracks apperently caused by the parasites contained debris of the injured hepatic cells , erythrocytes, neutrophils and a fre macrophages . At some places the headup tracks were surrounded by fibrus tissue. In the heald up tracks there were deposition of haemosiderin graunles as identified by the prussian Blue Reaction. The healed up tracks appeared as scars and the scarred areas were linked to the adjacent portal triads by means of finger like projections of the connective tissue . Sections of parasites were not encountered in the haemorrhagic tracks.The section also revealed severe congestion, the central vein and sinusoids being engorged with erythrocytes.There were infilteration with mononuclear cells( Fig.3) The portal tract and common bile duct were found to be sevrely affected.There was diffuse increase in fibruus tissue in the portal area. The bile duct in the portal area showed hyperplasi .The portal vein was dilated. The wall of the hepatic arterioles showed thickening due to hyper trophy of the median coat resulting in narrowing of the lumen. The portal triads appeared as wide areas of fibrous connective tissue surroundedby atrophied hepatic cells ( Fig. 4).

Fibrous bands from the portal tract were seen invading the liver parenchyma and were found to isolate islands of hepatic cells in their immediate vicinity.The hepatic cells showed atrophy and affected areas were in filterated with mono muclear cells.The microscopical picture indicated mono lobular and paricellular type of cirrhosis.

Sections of common bile duct showed sections of adult parasites firmly attached with the mucus membrane with its poterior sucker. A flask shaped projection of the mucosa was sucked in filling the cancavity of the large posterior sucker, (Fig. 5). The muscosa of the bile duct showed hypertrophy and hyper-plasia and its deeper layer contained many mucus secreting glands. The mucosa was infillterated with mononuclear cells. There was also pronounced hyper plasia and hyper trophy of the lining epithelium of mucosa of the bile duct. The proliferative changes led the epithelium to assume a papillomatous pattern.The surface at places showed necrosis and desquamation of the biliary epithelium. The necrotic area at places werex covered with in lamatory exudate (Fig.6) . The outer most layer showed pronounced proliferation of fibrous connective tissue infilterated with mononuclear cells which also infilterated the adjacent liver parenchyma ( Fig. 7) Numerous small arterioles were seen with greatly hypertrophied media and their lumina reduced. The smooth muscles were showing atrophy and they were separated by the proliferating fibrous tissue( Fig. 8) .

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Sections of the gall bladder showed atrophy of the muco a and its gladular epithelium for the most part absent.

## DISCUSSION

The general gross and histo pathological changes encountered in the present investigation was very much similar to th se described by Kulasiri and Seneviratne ( 1956) in fuf buffaloes.

It was appreciated during the present investigation that in order to under stand the pathogenesis encountered in the liver , it was quite essential to have a knowledge of the route of migration of the metacerecariae . Through the life cycle of the parasite, Gigantocotyle explanatum was studied by Singh(1958) but the author did not mention the route of migration of the metacercariae .But however Kulasiri and Seneviratne ( 1956) on the basis of the lesions encountered in the liver and because of the location of the parasites in the liver parenchyma, portal veins and bile ducts of buffaloes, suggested a transperitoneal( abdominal) route of migration of the metacercariae as in fasciola species ( shaw , 1932,Schumacher 1938, Morill and Shaw 1942,Krull and Jackson, 1943. )

During the present investigation the gross and histo pathological lesions were suggestive that the route of migration of the metacercariae might be transperitoneal (abdominal ) in goats like that of buffaloes as evidenced by the presence of haemorrhagic tracks made by the parasite during their

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migration and their subsequent healihg and replacement by fibrous tissue. But during the histopatholigical examination the preadult stage of the parasite was not encountered in the paranchyma or the portal vein as described by Kulasiri and Seneviratne ( 1956) in buffaloes . The parasites might have migrated to the bile duct to spend remainder of its life.

The pathological changes in the liver were always dominated by contant involvment of the bile duct.

The hyper plasia of the mucus membrane, increase in the number of mucus secreting cells and m necrosis of lining epithelium of the common bile duct might be due to the irritation caused by the suckers of the parasite. But hyper plastic changes encountered in the portal bile ducts could not be attributed due to the suckers or due to a mechanical irritation by the parasite ( Since the parasites do not possess any spines on their cuticle ) but might be due to the liberation of a diffusible chemical irritant or irritants. The muscular hypertrophy in the hepatic arterioles might be due to hepatic hyper-tension resulting from a diffusible chemical irritant or irritants as suggested by Kulasiri and Seneviratne ( 1956). The increase in connective tissue of the portal area and monolobular cirrhosis is expected to be due to widespread blocking of the bile ducts by the parasite.

One parasite was also encountered in the gall bladder, but the pathological picture does not suggest infection

at this site and ix xx as it is considered that the parasite might have migrated into the gall bladder after death of the host when the sphincter muscle at the mouth of gall bladder must have ruxxxx rel xed.

Hyper plastic changes in the bile duct epithelium similar to those encountered in the present investigation and attributed to the liberation of Chemical irritant by the parasites have been reported in fascioliasis( Dawes 1963 a) and Dicrocoeliasis (Dhar and Singh 1963 )

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# F A S C I O L I A S I S.

## I N T R O D U C T O N.

Voluminous work have been carried out on different aspects of Fascioliasis , but there is still a wide gap in our knowledge regarding the pathology of the de ease. Dawes and Hughes ( 1964) have rightly pointed out " that the pathology of fascioliasis is also a rather neglected subject about which much more information will be required before a reliable assessment can be made of the parts played by the parasite and host respectively". Most of the work onpathology of the desease have been carried out in small laboratory experimental animals and in fact very little work have been carried out on pathogenicity of Fasciola infection in goats.

## REVIEW OF LITERATURE

Fascioliasis as a separate entity was first recongnized by Jean de Brie ( 1379). Since then this condition have been reported from different parts of the world. As far as the author is aware perhaps in no part of the world the domesticated animals are free from the disease caused by Fasciolaspecies.

The enonomic loss caused by this parasite has been quite considerable. Thus according to Sen ( 1950) statistics show that in Germany in the year 1924 and 1925 there was so severe out breack of Fascioliasis amongst cattle, sheep and goats that 18,000 cattle, 60,000 sheep and 2,500 goats were

were distroyed and also caused the death of 18,000 cattle, 12,000 sheep and 1,200 goats. He observed similar incidence in ~~X~~ Yugoslavia during the year 1926-27 and during that period 62,000 cattle, 294864 sheep and 6,141 goats died due to Fascioliasis. In ~~X~~ India the decease had been reported for a long time from Hydrabad, Almora, Assam and from other parts.

Verma ( 1954) , on the basis of faecal examination of goats reported Fasciola infection from Supaul, Saharsa, and Purnea area of Bihar State. He reported that ~~x~~ 78% of goats in Supaul, 71% in Saharsa and 50% of goats in Purnea were infected with fascioliasis. During his survey in determining the prevalent species of Fasciola in those area, he found that it was a new species of the F. indica( Verma , 1953) quite distinguishable from F. hepatica ~~and~~x or F. gigantica .

Lungn( 1959) reported that before 1955 losses of sheep due to liver fluke infection were small in Rumania but during the subsequent rainy and winter season there were acute out breaks of infection in may areas. It was estimated that 18% of stock were lost in 1956. Mirzaev ( 1959) reported that on the collective farm "Pobeda " 120 sheep were lost due to fascioliasis in 1956 and 135 in 1957. Sarwar( 1960) during survey of the helminths of sheep and goats carried out in Kharan, Chagai and Kalat reported that in Kalat ( which includes Jhalawan and Sarawan ) liver fluke infection in Sarawan is due to F. hepatica but in Jhalawan it is caused by F. gigantica

Bhatia (1960) reported about the liver affections with three species of flukes parasitizing Indian sheep. Popov and Kalatina (1962) reported the incidence of Fasciola in 1,197 of 3,483 sheep in Central Cancasus. Balasingham, (1962) reported the incidence of Fasciola gigantica in locally bred goats as 2.25% which has increased markedly in part from years in Singapore island.

Regarding the patholigical features of fascioliasis Lutz( 1893) was first to initiate work on experimental fascioliasi-s. He fed encysted metacercariae of Fasciola gigantica to goats guineapigs, rabbits and a rat. During the experiment he believed that theyoung forms of the parasites were carried passively to the liver by way of hepatic portal system. This idea was supported by other workeers notably Railliet et al ( 1913), Compes ( 1923) , Noller ( 1925), Noller and Schmid ( 1928) , Mareck ( 1927) Bugge ( 1935) and others.

Sinitsim ( 1914) observed a large number of flukes creeping over the viscera of the abdominal cavity and then entering the liver.Shirai( 1927) fed encysted metacercariae to guineapigs and recovered flukes from the bile ducts. Shaw ( 1932) observed that the route of migration of young flukes in the final host was by way of the peritoneal cavity from the intestine to the liver. Stewart ( 1934) observed during an out breack of acute liver rot in sheep accompanied by suddenx death which on post -mortem revealed that the capsule was studded with darker areas, measuring about 3 m.m. across

which were the points of entry of the cercariae. He also found large number of flukes in the liver. Sivitil ( 1934) discribed pathological changes in liver infected with Fasciola henatica . He found that in majority of cases the bile ducts of he left lobe only showed lesions . He also described that early stages of infection the mucus membrane of the bile ducts were swollen their lumina were enlarged and walls were infilterated with celluiar elements especially with leucocytes. In later stages there was hypertrophy of fibrous tissue accompanied by regressive and dystrophic changes with frequent calcification of the walls of the bile duct. Bugge ( 1935) reported that in early days it was believed that the young flukes made their way to the liver via the bile duct, later on it was held that they actively penetrated the wall of the intestine, traversed the abdominal cavity and pierced the capsule of the liver but during his observation the author showed that they generally genera ly make their way to the liver via the postal circulation. Lavier et al ( 1938) observed carcinomatous lesions associated with Fasciola dn Dicrocoelium infection of the sheep and concluded that Dicrocoelium re more like to have been cancer producing than the Fasciola . Morill and Shaw ( 1942) discribed incattle the presence of focal subcapsular lesions in the liver due to fascioliasis and suggested that young flukes reached the liver by penetrating through the capsule and parenchyma. Krull and Jackson(1943)

fed mice and guineapigs with Fasciola cysts and recovered flukes from the abdominal cavity and liver. Flukes thus recovered were readministered into abdominal and pleural cavity of sheep, rabbits and guineapigs . The authors suggested that the guineapigs were unsatisfactory hosts because the flukes could not get mature in them. Their experiment also established that when flukes were transfered to pleural cavity of rabbits they might enter the lungs or penetrate the diaphragm into the abdominal cavity and enter the liver.

Bonciu et al (1954) observed three main types of histopathological changes in the liver of guineapigs which died of Fasciola infection (i) enlargement of the liver with degenerative, necrotic lesions and fatty degeneration (ii) hyper-plasia and sclerosis of connective tissue and lastly (iii) a small liver with hyper-plasia of the connective tissue. In addition they also observed chronic cholangits, pericholangitis and hyper plasia of blood, lymph and bile vessels.

Sogoyan ( 1955) described in the patholigical changes in liver of sheep experimentally infected with Fasciola gigantica . He found that nearly all the flukes migrated to the liver via the blood stream and only small number of them actively peneterated the intestinal wall and entered the abdominal cavity. He also

described that in acute cases the migrating fluke caused traumatic hepatitis with destruction of liver cells and haemorrhage where as in chronic cases when the flukes have peneterated the bile ducts, proliferation of fibrous tissue and slight regeneration of the liver parenchyma was observed. Sogoyan ( 1956) described the comparison of the pathological changes in sheep caused by Fasciola hepatica and Fasciola gigantica . He observed that the damage caused by the later species were much more than the former species. Lapage ( 1956) described that after ingestion of the metacercariae the cercariae scapes from the cyst into the duodenum and penetrate into the deodenal wall. Some of the flukes after penetration reach the liver via the blood stream or by crawling up the bile duct but most of them migrate to the liver via the peritoneal cavity after penetrating the intestinal wall. Then the parasites enter the liver by penetrating the capsule. After enteringkxinto the liver parenchyma they grow and wander for about a month and then reaches the bile duct.

The young flukes while wandering in the liver parenchyma destroy the later leaving haemorrhagic tracks ,when they reach the bile duct they irritate biliary mucosa by their cuticular spines and leads to chronic cholangitis and cirrhosis. Urquhart ( 1956) studied the gross and histo- pathology of experimental fascioliasis in rabbits. Grossly on the surface of the liver there were

were cream coloured or pink lesions measuring 1-2m.m. in diameter or as streaks up to 10 m.m. in length and about 2 m.m. wide. Microscopically it was shown that those areas were necrotic tracts caused by the migration of the fluke in the liver parenchyma. The spaces left by the fluke were filled with cell debris, neutrophils, lymphocyes and red corpuscles. The column of liver cells adjacent to the tract were atrophied, the nuclei were pyknotic and the sinusoides in his area were distended with large mono nuclear cells, lymphocytes and granulocytes of which mono nuclear cells predominated . The bile ducts were frequently damaged by the parasites. Rupture of the bile duct. During migrating period particularly in older infestation in-farcts were commonly observed . Cvjetanovic et al ( 1958) described the patholigical changes in the liver in Infectious necrotic hepatitis activated by fluke infection in sheep. Post mortem findings included congestion and yellowish grey di colouration of liver with fluke migration tracks clearly visible, and Cl. oedimatiens was regarded as the primary cause of death. Gavez and Maglajlic ( 1959) described a case of carcinoma from bovine in association with Fasciola hepatica cirrhosis but it was not confirmed that whether carcinoma was due to heavy fluke infection or to some other factor.

Dawes ( 1961c) stated that the young flukes are tissue feeders and Sinclair ( 1962) confirmed the findings of Dawes . Gresham and Jennings ( 1962) observed that in early stages of Fascioliasis there were haemorrhagic

tracks through out the liver surface and focal areas of fibrinous peritonitis resulting from the migrating flukes. They also observed a chronic cholangitis and occulusion of smaller bile ducts.

Katiyar and Tewari ( 1962) described the pathological changes in acute fascioliasis amongst sheep in Kumaon hills. They observed on Post- mortem examination that the liver was congested, enlarged haemorrhagic and friable and showed numerous punctures on the peritoneal coverings through which the flukes ( Fasciola giganti-ca) were protruding out. Some of the livers usually contained numerous cavities filled with dirty fluid and young flukes. On histopatholigal examination the section of liver showed areas of haemorrhages corresponding to the tracts made by the migrating flukes wit marked parenchymatous cell destruction and leucocytic in-filteration. Parasites surrounded by haemorrhagic exudates ere also present. In later stages there were varying amount of fibrous tissue distributed around one or more lobules of liver tissue particularly in neighbour hood of bile ducts.

Osborne ( 1962) described the pathology of Fascioliasis and black disease in sheep in New England .He observed on autopsy that most cases were characterised by anaemia and emaciation and liver damage with flukes , consisted of diffuse fibrosis and haemorrhage involving the whole organ. Bleeding pits were commonly present in the

capsule with portions of immature flukes protruding . In many cases caseous lesions were present whichwere not encapsulated and were no surrounded by any marked zone of reaction. The author also described that in infectious necrotic hepatitis the liver showed necrotic lesions with xx their surrounding zone of reaction and the slight haemorrhagic lesions produced byvery small flukes in small numbers. Dawes ( 1963 a, b) observed that during early stages of infection in mice with Fasciola - hepatica the young flukes feed e clusively on hepatic cells which has been broken down by oral sucker and pharynx into a homogenate containingx Kupffer cells, invading leucocytes and some erythrcytes . He further described that adult flukes in the bile duct of verteberate hosts sustain the tissue feeding habbit. When immature flukes are invading the liver parenchyma the biliary epithelium show hyperplasia and the under lying connecitive tissue show fibrosis.The author concluded that the flukes enter the bile duct when continued feeding on hepatic cells would greatly endanger the life of the host and due to inflamatory reaction the flukes are provided there with a"pasture" of tissue on which to feed.

Smith and Jones ( 1958) described that infective metacercariae of F. hepatica upon ingestion by a definitive host penetrate the intestinal wall and migrate to the liver parenchyma via the peritoneal cavity . After migrating into the liver parenchyma the fluke

enter the bile duct where it spends its adult life. The parasites in the bile duct irritate the mucosa and as a result the biliary epithielium is stimulated to excessive growth in places and is erroded in others. They further described that the walls of the bile ducts become greatly thickend from fibrous proliferation. The fibrosis around the bile ducts extends deep into the hepatic lobules producing severe perilobular fibrosis.

Thorpe ( 1965 during his studies on the pathology of experimental fascioliasis in the albino rats suggested that there was transperitoneal infection of the liver and discussed the gross and histopatholigical changes in the liver.

## RESULTS

### Gross Pathology:-

The capsule was thickened. The liver was congested. The surface of the liver was studded with dark haemorrhagic spots probably indicating the entrance of the fluke into the paranchyma ( Fig. 9) . When these dark spots were followed into the parenchyma numerous immature flukes, bathed in turbid blood tinged fluid containing tissue debris were seen. In one case the liver was very tough and friable . The capsule was thick, white and glistening. The surface of the liver showed irregular depressions and elevations indicating

atrophy ( Fig. 10). The gall bladder was distended with thick bile containi g tissue debris . When a deep incision was given into the liver , numerous haemorrhagic spots and tracks containing flukes were seen ( Fig. 11). In one case a big blood clot surrounded by a thick fibrous tissue capsule was seen. The distended bile ducts with their thick walls containing liver flukes were seen ( Fig. 11,12). When the bile duct was opened blood tinged fluid containing tissue debris oozed out. The liver parenchyma at places were studded with yellowish brown pigments.

Histo Pathology:-

The capsular thickening was composed of a laminated collagenous membrane and fibroblastic layer. Several haemorrhagic tracts and spots of different sizes were seen ( Fig. 13). Within the haemorrhagic tract sections of para its surrounded by haemorrhagic exudate consisting of leucocytes erythrocytes and degenerated hepatic cells were seen (Fig.14). The leucocyte consisted of neutrophils, mononuclear cells and occasional eosinophils .(Fig.15) . The hepatic cells in the immediate vicinity of the haemorrhagic tract were deeply stainedx and showed necrotic changes with leucocytic infilteration comprising neutrophils, eosinophils and mononuclear cells. The haemorrhagic tract which were left by the parasites were surrounded by moderate amount of fibrous connective tissue indicating start of healing(Fig.13)

Hepatic cells in the immediate vicinity were showing atrophy. Older haemorrhagic tract appeared as scar infilterated with mononuclear cells. At places scars fused with each other and with the portal area giving the picture of irregular fibrosis (Fig. 16). The scars were studded with yellowish brown haemosiderin granules which were identified by Prussian Blue Reaction as blue granules. The liver showed monolobular cirrhosis with newly formed bile ducts in the portal area(Fig.17) In few cases extensive coagulative necrosis and caseation necrosis were seen. The area was infilterated with leucocytes containing mostly neutrophils and few mononuclear cells(Fig.18) In one case a blood clot surrounded by thick fibrous tissue capsule was seen. The hepatic cells in the immediate vicinity was showing atrophy. In the portal area the wall of the hepatic arterioles were thickened due to medial hypertrophy. The mucus membrane of the bile duct was showing hyperplastic and hypertrophic changes.There were increasedmm number of mucus secreting glands. The biliary mucosa was showing papilla like projection due to proliferative changes the tips of which were showing necrosis and desquamation. The mucus membrane was infilterated with leucocytes comprising mostly monunuclear cells, the outer most layer of which showed proliferation of fibrous & tissue ( Fig. 19). The lumen of the bile duct revealed mature flukes inside it. In one case the lumen of the bile duct contained eggs of the Fasciola ( Fig. 20).

## D I S C U S S I O N

The gross and histo pathological changes in the liver of goats particularly the presence of haemorrhagic spots on the surface of the liver leading to haemorrhagic tracts were quite akin with the observations made by the previous authors in sheep notably Sogoyan ( 1955), Osborne ( 1962) and Katiyar and Tewary ( 1962) and in goats by Sugiura ( 1954).

Similar types of lesions were observed in small experimental laboratory animals by Shaw ( 1932) Schumachar(1938) Morill and Shaw ( 1942) ,Dawes(1961a) , Thorpe ( 1965) and many others.

As described by previous authors namely Urquhart ( 1956), Dawes ( 1961 b), Taylor ( 1964) and Thorpe ( 1965) the cellular content of the track comprised of large numberof Red Blood corpuscles , degenerated hepatic cells, neutrophils, mononuclear cells and occasional eosinophils. But the multinuclear giant cells on the border of the tracks described by those authors in addition were not observed during the present investigation.

During the present investigation such types of lesions were suggestive of transperitoneal migration of parasite and this was in complete agreement with the observations made by the previous authors in small laboratory animals notably Shaw ( 1932), Schumachar ( 1938) , Morill and Shaw(1942),

Dawes ( 1961a) , Thorpe ( 1965) and many others.

The hepatic cells bordering the periphery of the tracks occasionally showed an increased staining affinity to h emotoxyline, Dawes ( 1961 b) postulated that this peculiar staining character might be due to the alterations in the cytoplasm probably enzymes of the cells, from the materials derived from the caeca of the parasites during defecation.

It is well known that reparir of any damage to the hepatic parenchyma is brought about by a process of regeneration from the existing hepatic cells in its neighbourhood . In the present investigation nonm of the tracks manifested any regenerative activity in neighbouring hepatic cells but the repair was brought about by a process of substitution by connective tissue elements. Therefore it can be postiulated that some factors or factors are at play which has inhibited the regenerative capacity of the hepatic cells and at the same time allowed the connective tissue elements to proliferate. It is a known fact that retardation of regeneration of hepatic cells are brought about by interference with hepatic circulation by partial obstruction of vein and retention of the bile. In the present investigation increase in the periportal connective tissue, thickening of the hepatic arterioles leading to reduction of the lumen and partial obstruction to the flow of bile by the parasites noticed may be incriminated as possible factors which has

inhibited the regeneration of the hepatic cells. However this aspect needs further experimental elucidation.

Extensive coagulative necrosis in two of the specimens are in close agreement with the lesions of black disease and Infectious necrotic hepatitis described by Cvjetanovic et al ( 1958), Lapage ( 1965), Osborne ( 1962) and Runnels et al ( 1965) in lambs, but during the present investigation in none of the two specimens clostridial organisms ( which might be few in number and escaped detection under the microscope ) could be seen in the sections.

The coagulative necrosis might also be due to prolonged action of some chemical substance or substances liberated by the flukes. However this aspect needs further experimental elucidation.

Lavier et al ( 1938) reported occurrence of carcinoma in association with fasciola and Dicrocoelium in festation of the liver in a sheep. Gavez and Maglajlic (1959) also reported occurrence of Carcinoma in cattle in association with fascioliasis . But in present studies only hyperplastic epithelium of the bile duct showing no neoplastic tendencies were observed.

The vascular changes encountered inxthe present studies consisted of hypertrophy of the arterial wall situated in portal tract. These changes and hyperplastic changes encountered in the bile duct, increase in periductal

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connective tissue and the increase in the connective tissue in the portal areas leading to monolubular cirrhosis might be due to a diffusible chemical irritant or irritants elaborated by the parasites. The changes encountered in the bile duct and monolobular cirrhosis in the liver might also be due to irritation of the mucosa by the cuticular spines of the parasite.

Hyper-plastic changes in the bile duct epithelium similar to those encountered in the present investigation and attributed to the liberation of a possible chemcial irritant by the parasite have been reported in fascioliasis ( Sugiura, 1960, Dawes , 1963c ) . Whereas Lapage ( 1956 ) , Runnels et al ( 1965) and Jubb and Kennedy ( 1963) have reported that these changes are due to constant irritation by the cuticular spines of the parasite.

Nara ( 1959) has shown that administration of irritant substances like dimethyle formamide and Nishimoto ( 1959) has hsown that administration of Cadmium sulphate are capable of inducing cirrhosis of liver.

In the opinion of the author the biliary changes and cirrhosis of the liver encountered in the present investigation might be due to the constant irritation by the cuticular spines of the parasites which is further aggrevated by the liberation of possible chemical irritant or irritants by the parasites.

Lutz ( 1893), Bugge ( 1935) , Sogoyan ( 1955) in their experimental studies on fascioliasis postulated that the flukes often reached the liver via portalcirculation. But during the present investigation there were no evidence of entry of the flukes in the portal vessels during their migration in liver and this was in complete agreement with the observations made by Dawes ( 1961 b). However this aspect needs further experimental elucidation.

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## HYDATIDIOSIS IN LIVER

## I N T R O D U C T I O N

Echinococcus granulosus , one of the smallest tape worms is most commonly found in small intestine of dogs, fox and several wild carnivores. The immature stage of this parasite is known as Echinococcus or Hydatid cyst.The intermediate hosts of this parasite are man, sheep, goat and allt domestic mammals, and gives rise to typical hydatid disease in the intermediate hosts. The incidence of hyd tid disease inhuman in any country is closely related to the incidence of the disease in domesticated animals.

## REVIEW OF LITERATURE

Sami ( 1938) reported that at D. Ghazikhan (W.Pakistan) 1% of sheep and goat suffered from hydatid disease. Maqsood (1946) reported that at Lahore 66.1% of cattle and 88.8% of buffaloes harb ured hydatid c t.Chhuttani and Chugh(1957) reported that at Patiala the incidence of hydatid disease was 2.8% amongst sheep and goat. Buljevic and Sibalic ( 1958) reported that in Pancevo ( Yugoslavia ) during 1948 to 1956 the incidence of hydatid disease amongst sheep was 95%, amonst cattle 78.4% and amongst pi s 34.8%. Verster ( 1962) reported an average incidence of hydatidm disease in south Africa as 1.798% amongst sheep and goat, 0.633 % amongst cattle. Thornton ( 1962) reported incidence of hydatid disease

amongst sheep in France- 0.03% , Italy -15-52% , Spain-28-40.6% Algeria 21.8 -95%, Kenya -53%, Yugoslavia -35-72% Turkey -4%, Middle East 4.6-41.4% Australia-36%, Newzealand-36% , Argentina 11%.

Sharma and Chitkara ( 1963 ) reported incidence of Hydatid disease in Amritsar as 3.5% amongst sheep and goat of which 33.3% of animals harboured cysts in lungs 23% in liver and 43.6% in lungs and liver.

Pandey( 1966) reported incidence of hydatid disease in goats in the city of Patna as 6.5% of which 2.7% of goats showed liver affections.

The exact incidence of hydatid disease in goats in this country as a whole is not known. Previous workers in India have not worked out the incidence separately for sheep and goat.

Though the incidence of hydatid disease in domesticated animals is high, it appears that very little wor have been done to detail the changes encountered in various organs affected by these cysts, especially in goats. Yamashita et al ( 1956) have given an account of the histpathological changes of hydatidiosis in 4 sheep secrificed at different intervals after an experimental infection with the ova of parasites. Yamashita et al ( 1956a) have also given an account of he histopathological changes of hydatidiosis in two natural cases in sheep.

Nosikx and Pustover ( 1959) have recorded the pathological reaction ofthe host towards the cyst. In those cases authors described that there was a fibrofilamentous connective tissue capsule around the cyst. Where the reaction was severe there was anx accumulation of histiocytes around the cyst. While the parasites showed the formation of daughter cysts. In very severe reaction the parasites were seen to under go resorptive changes.

Ahluwalia ( 1960) studied the histopathological changes encountered around the cyst in pigs and reported that around the cyst there were fibroblasts arranged in perpendicular fashion ,these fibres in turn were surrounded by mature collagenous fibrous tissue infilterated by macrophages, lymphocytes and eosinophils.

Mortelli and Gabbanini ( 1959) described histpathology of portal lympt node incattle and sheep with heavy Echinococcus infestation. Ihey discribed that portal lymph nodes presented a chronic inflamatory process. Characterised byd diffuse reticulo endotheliosis and fibrosis of the sinuses , M Plasma cell infilteration, hyperplasia and hypertrophy of smooth muscles, atrophy of the follicles and hyaline degeration of connective tissue.

Pandey ( 1966) studied the gross and histo pathological changes encountered in the liver of goats infested by the cyst . He reported that the cyst wall was composed of a thin

inner nucleated germinal layer of embryonal cells , surrounding this there was a thick outer laminated layer of faintly stained hyaline connective tissue. In fertile cysts brood capsules with scolices inside it, were attached to the germinal layer . The cyst wall proper was surrounded by an outer capsule of dense fibrous connective tissue formed as a result of destruction of liver cells and subsequent replacement by fibroblasts. The changes in the liver parenchyma were of degenerative and proliferative nature. He also described pericellular, monolobular and central cirrhosis in the liver.

Sengupta ( 1966) studied the gross and histopathological changes in the liver of buffaloe infested with the cyst. He reported that in early stage the cystic structures from within outwards revealed germinal layer surrounded by thin cyst wall. These structures were surrounded by connective tissue capsule arranged parellel to the parasitic wall. At the junction of the connective tissue capsule and the hepatic parenchyma there were small number of mononuclear cells. The hepatic cells in the immediate vicinity of the connective tissue capsule showed atrophy.

In advance stage germinal layer from all the cyst were absent. Parasitic cyst wall was either disorganised or disappeared where the parasitic cyst wall was present it showed degenerative changes. These structures were seen surrounded by a zone of epitheloid cells interspersed

with a few fiant cells of foreign body type.These areas in turn were surrounded by a connective tissue capsule infilteratéd by a large number of small mononuclear cells.

## R E S U L T

For the sake of convenience the lesions of hydatidiosis is described according to the age of the cyst and their tissue reaction ,under two headings viz.

(i) Early stage (ii) Advance stage

(i) Early stage:

Gross Pathology:-

The parenchyma was studded with cysts varying in size and number . The size varied from a marble to tennis ball and number varied from one to numerous(Fig.21) The wall of thecysts were hard and had a tendency to collapse on presure. When the cysts were incised it manifested anm empty cavity lined by a smooth membrane which could be detached out. The cysts were filled with clear watery fluid. All the cysts were of unilocular variety. The parenchyma arround the cyst showed congestion. In case of fertile cyst the smooth membrane wasx studded with granular structure which on microscopical examination proved to be brood capsules.

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Histopathology :-

Microscopically the focus was characterised by the cyst walland changes around it. The cyst wall was composed of an inner single layered nucleated germinal layer of embryonal cells. Surrounding this layer there was a thick outer concentrically laminated layer of faintly stained hyaline connective tissue ( Fig. 22) . In fertile cysts brood capsules with scolices inside it were attached to the germinal layer (Fig. 22)The cyst wall proper w as surrounded by an outer dense fibrous connective tissue capsule formed as a result of destruction of liver cells and subsequent replacement by fibroblasts. The capsule had an inner zone of mature connective tissue fibres with few nuclei and an outer , more cellular zone of newly formed fibrolblasts. In some of the specimens the conrective tissue was showing hyalinization. The arrangement of the fibroblasts were parellel to the cyst wall. At the junction of the hepatic parenchyma and the fibrous tissue capsule there were infilteration with mononuclear cells (Fig. 23). Islands of hepatic cells and newly formed bile ducts were encountered amidst the fibrous tissue capsule. The hepatic cells in the immediate vicinity of the connective tissue capsule showed atrophy and their nucleus were showing pyknosis, kryarrhexis and karyolysis indicating on set of necrosis . The hepatic cells farther away showed degenerative and proliferative changes. There were pericellular and monolobular cirrhosis (Fig. 24, 25).

The architecture of hepatic cords were disturbed. The central vein were no more central and in areas of heavy infection the hepatic cords looked like island between cystic structures. The portal area as a whole showed proliferation of fibroblasts and had many newly formed bile ducts. The central vein and sinusodis were engorged with blood.

(ii) Advance stage

Gross Pathology :-

Macroscopically the cysts were yellowish grey in colour and hard to feel. When the cysts were incised the content appeared clotted and in few cases the cysts appeared to be filled up with caseous material.

Histopathology:-

Microscopically the germinal layer from all the cysts were absent. Parasitic cysts wall was either disorganized or disappeared. Where the parasitic cyst wall was present it showed loss of its laminations(Fig. 26) . The cystic content and its associated structures appeared as caseonecrotic mass taking an intense eosinophilic stain. These structures were surrounded by a zone of mononuclear cells interspersed with a few giant cell of foreign body type and occasional eosinophils ( Fig. 27) These areas in turn were surrounded by a thick connective tissue cpx capsule infilterated by a large number of mononuclear cells.

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Islands of hepatic cells and newly formed bile ducts were seen amidst the connective tissuexx capsule.

## D I S C U S S I O N

In order to understand the developement of the cysts and the tissue reaction encountered in it, it is nece sary to have a knowledge of the life cycle of the parasite leading to the developement of the cysts in various organs. In breif the gravid segment of the parasite T. echinococcus is ingested by any susceptible host, the hexacanth embryo passes through the intestinal wall and are carried to the liver by portal vein. The larger embryos are arrested in the liver and the smaller ones pass through the the liver cappilaries and reach the lungs, while those embryos which succeed in passing the lung cappilaries , enter the systemic circulation to reach the v ri us organs of the body.

Jubb and Kennedy ( 1963) have reported that sheep is the only normal intermediate host for T. echinococcus and in cattle 90% of the cysts are sterile. In the present investigation 75% of the cysts were found fertile. Yamashita et al ( 1956) described necrosis of the hepatic cells in the vicinity of the cysts. In the presenct investigation the nuclei of the hepatic cells in the immediate vicinity ofthe cysts were showing pyknosis, karyorrhexis and karxyolysis indicating necrosis and thecells were showing

atrophy . The authors h ve described that the necrotic changes are due to heavy pressure exerted by the cyst. But it can not be ascertained that necrotic changed are either due to pressure or due to metabolites diffusing outside the cyst wall. However this aspect needs further experimental elucidation.

Ahluwalia ( 1960) has reported h that fibroblasts in the immediate vicinity of the cyst wall are arranged in perpendicular fashion , but during the present investigation in non of the specimens similar arrangement of the fibroblasts were encountered.

It has been reported that in number of parasitic infections eosinophils are found around the parasite . However in the present enquiry the predominating cells were mononuclears with occasional eosinophils in early stage and monomuclear cells interspersed with gian cells with occasional eosinophils in advance stage. Mac Faulum (1962) has reported that eosinophils appear in situation where antigen antibody reaction are taking place. In the present investigation only occasional eosinophils were encountered, it could thus imply that the antibody contents in the body have not reached sufficient concen eration to elicit a reaction purely made up of eosinophils.

Smith and Jones ( 1958) described that dense fibrous tissue capsule with little cellular reaction

occured arround more mature cyst. In the present enquiry such lesions were only seen arround the cyst in early stage. This variation might be due to species susceptibili-ty. The authors also described marked eosinophilic infilteration arround the cyst in early stage. But the during the present investigation in goat liver, only occasional eosinophils were encountered arround the cyst wall in early stage and advance stage. The predominating cells were mononuclears.

During the present investigation marked pericellular and monolobular cirrhosis were encountered in the specimens having numerous cysts.

The parasitic cyst appear to release some substance after death or during their disintegration which may be responsible for the nature of cellular reaction around the cyst. In early stage only fibrous tissue capsule infilterated with mononuclear cells were present around the cyst wall and in advance stages mononuclear cells interspersed with giant cell of foreign body type were present around the cyst wall .

Pericellular cirrhosis and monolobular cirrhosis in the specimens having numerous cysts might be attributed due to heavy pressure exerted by the cysts or might be due to certain substance diffusing from the cyst acting as mild irritant to the liver tissue.

Thus the histological structure varied according to the number, age and stages of development of the cysts. The histological appearance of the lesions during the present investigation were highly suggestive of a proliferative inflamation.

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# C O N G E S T I O N

## I N T R O D U C T I O N

It is well known that the activity of the organ is responsible for the blood supply to any organ or tissues. A regulated ammount of blood supply to various organs is controlled by means of constriction or the dilation of the blood vessels. When there is any obstruction on the venous side, it results in to passive congestion of various organs due to diminished out-flow of blood from those organs. Smith nd Jones ( 1958) , Runnels et al ( 1965) have reported that domestic animals frequently suffer from passive congestion of liver.

## RESULT

Gross pathology:-

The organs were dark red in colour and when the liver was cut large quantities of purple coloured blood oozed out. On the surface of the liver there was fine sprinkling of dark brown and grey reminisent of the outside of a nutmeg. Thus it gave a typical character of "Nutmeg" liver. The organs were enlarged of dark brown clour and presented a mottled appearance ( Fig. 28).

Histopathology:-

Central vein and sinusoids surrounding them were engorged with blood. The hepatic cells were showing various degrees of fatty changes.The hepatic cells were showing atrophy ( Fig. 29). The nucleus of the hepatic cells in the centrolobular region were showing karyorrhexis and karyolysis, .Fibrous proliferation was also encountered around the central vein.

## DISCUSSION.

Gaiger and Davies ( 1957), Smith and Joines ( (1963) Runnels et al ( 1965) have postulated that chronic veinous congestion of the liver results from stenosis or incompetence of the mitral or tricuspid valves of heart, but in animals the ricuspid stenosis or in-sufficiency is more frequent.They have also postulated that Chronic veinous congestion of liver is also followed by pneumonia, emphysema or interstial pneumonia which hinder the flow of the blood from the right ventricle which results into accumulation of blood in the posterior vena- cava and its branches.

Runnes et al have also postulated that myocardial failure, anomalies of the heart, constrictive lesions of the pericardium and epicardium are also possible cause of chronic veinous congestion.

In the present investigation the cause of chronic veinous congestion encountered in the liver could not be established.

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## ICTERUS or JAUNDICE

### I N T R O D U C T I O N

The term icterus or Jaundice is used to indicate the staining of certain body fluids and tissues with bile pigments as a result of their presence in the circulation. The pigments are bilirubin and its oxidation product biliverdin.

### REVIEW OF LITERATURE

Cordy and G wan ( 1956) studied the pathology of massive liver necrosis in sheep. The authors observed icterus in 50% of the cases, the liver was deep yellow to yellowish brown in colour with red or reddish brown foci which often coalesced. Microscopically the essential liver lesion was necrosis.They postulated that nutritional deficiency may be the cause. Mores et al ( 1957) studied the pathology of experimental ovine leptospirosis and observed icterus throughuut the body. They postulated that leptospira contain a haemolytic endotoxin or a substance which in the presence of specific antibody and complement will lyse erythrocyts in vivo and result into icterus .Bracewell ( 1958) gave a note on Jaundice in housed sheep . The author observed enlargement of liver and the colour of the liver was yellow ochre. Histologically he observed centrilobular or diffuse necrosis of the liver cells, distortion of liver cells columns and constriction of sinusoids. Smith and Jones(1958)

have classified jaundice on the basis of etiology into three types (i) Haemolytic (ii) Toxic and (iii) Obstructive. They have described that some haemolytic toxin, bacteria and certain blood prtozoa are responsible for haemolytic jaundice, some hepatotoxic substance either chemical or of plant origin are responsible for toxic jaundice and some obstruction in the bile passages are responsible for obstructive jaundice. They have also described th t in toxic jaundic the the essential liver lesion is characterised by liver necrosis. Mac Farlane et al ( 1959) studied the pathogenesis of facial eczema in sheep and observed that there is an acute calangitis which is succeeded by occlusion of the bile duct by fibrous tissue. This is followed by necrosis and icterus. Tustin et al ( 1960) studied the pathology of bacterial icterus in sheep and observed that there is generalised icterus and focal necrosis in liver. Gram negative bacteria was isolated . Khoteev ( 1960) studied the pathology of Lupin poisoning in horse and sheep. He observed that in acute poisoning the liver was yellow with red spots and in chronic poisoning the liver was flabby, slightly enlarged , yellowish and oedematous . Sharma (1961) studied the pathology of lantana toxicity in sheep and noted icterus, intense congestion of central vein, disorganisation of structure of hepatic cords around the central vein and dissociation of hepatic cells. Denz and Hanger ( 1961) observed that Myoporum lactum poisoning leads to liver damage, icterus and photosensitivity.

Seawright ( 1963) studied on experimental intoxication of sheep with Lantana camara and described that the pathological findings in sheep that died or were killed in moribund condition varied in accordance with factors leading to death of the animal. Where illness was of shorter duration ( about 12-14 days ) the liver was found to be bile stained and gall bladder greatly distended with pale green bile. Acute colangitis was occasionally found. Yellow bile stained livers showed no significant histological change except accumulation of bile pigment. Where the swelling of theliver was sufficiently prolonged sporadic atrophy and necrosis of cen ral zonal parenchymal cells was evident. Reddy ( 1964) studied on experimental intoxication of sheep with Lantana camara and discribed that the liver was enlarged ,swollen, congested and icteric and in some animals lobular markings of the liver was also present. Microscopically liver revealed acute and subacute toxic hepatitis a characterised by diffuse necrosis. In none of the cases he observed accumulation of bule pigment in the liver.

Absalyamov et al ( 1965) studied the pathology of haemolytic jaundicein Karakul sheep. They described that the liver showed megalocytosis, eosinophilic inclusions, distrophy and necrosis of liver cells, proliferation of epithelium of bile duct with formation of new ducts.

Senf and Seffner ( 1965) studied the pathology of Mercurialis annua poisoning in sheep and described that the principal post-mortem findings were degeneration of liver and icterus. Jha and Iyer ( 1966) studied the pathology of photosensitisation caused by experimental phenothiazine intoxication in cattle and sheep and described that liver appeared paler than normal and in some was depp yellow in colour.

Kiesel and Alexander ( 1966) studied the elctropheretic pattern in adult sheep on a magnesium deficient diet and described that six sheep developed icterus and post- mortem examination revealed liver and kidney damage.

Pienaar et al ( 1966) observed eosinophilic intranuclear inclusion in hepatocytes in enzootic jaundice in sheep in 24.3% of cases examined. They also discussed the possible nature and significance of the inclusiion and reached on a conclusion that they are probably of non viral origin. They also described that the lesions were similar to chronic pyrrolizidine alkaloid poisoning and chronic aflatoxicosis .

## R E S U L T

Gross Pathology:-

The liver was enlarged and was of dirty

yellow colour. The liver was severely congested in patches. On incision congestion followed about1-2 m.m. deep into the liver substance from the capsule. The lobular markings were prominent.

Histopathology:-

Microscapically the hepatic cords were showing disorganisation and hepatic cells were showing individualization which is an indication of onset of necrosis(Smith and Jones 1958) . The bile duct in the portal tract contained small amount of exudate containing few leucocytes and fibrin. The area in the vicinity of the capsule was severely engorged with erythrocytes. Sections did not reveal the presence of bile pigment.

## D I S C U S S I O N

During the present investigation disorganisation of hepatic cord structure and individualization of hepatic cells were noticed which is in close agreement with the findings of Sharma ( 1963) .

In the present investigation the type of jaundice could not be established. Various etiological factors have been incriminated by diff rent authors, but during the present investigation the cause of icterus could not be established which needs further experimental elucidation.

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yellow colour. The liver was severely congested in patches. On incision congestion followed about1-2 m.m. deep into the liver substance from the capsule. The lobular markings were prominent.

Histopathology:-

Microscapically the hepatic cords were showing disorganisation and hepatic cells were showing individualization which is an indication of onset of necrosis(Smith and Jones 1958) . The bile duct in the portal tract contained small amount of exudate containing few leucocytes and fibrin. The area in the vicinity of the capsule was severely engorged with erythrocytes. Sections did not reveal the presence of bile pigment.

## D I S C U S S I O N

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# N E C R O S I S

## I N T R O D U C T I O N

Local death of tissue within the living body is known as necrosis. Liver is such an organ that it is very prone to necrosis especially due to toxins. As described in the introduction chapter liver detoxifies circulating toxins and when the liver does not cope with, death of the liver cells takes place. According to Smith and Jones ( 1958) , from stand point of location, necrosis in the liver may take any one of the five forms;(i) Diffuse necrosis(ii) Focal necrosis (iii) Peripheral necrosis (iv)Midzonal necrosis (v) Centrilobular necrosis ( vi) Paracentral necrosis.

## REVIEW OF LITERATURE

Stepkowski and Woloszyn ( 1956) studied the pathology of Bradshot and on post-mortem examination they found enlargement, pallor, and friability of the liver with small areas of necrosis , they recovered Cl.oedimatiens from theliver. Haris et al ( 1957) studied the pathology of Senecio longilobus poisoning in sheep, microscopic examinations of liver revealed fatty metamorphosis,necrosis and fibrosis . Brock et al ( 1957) described the pathology of chlorinated naphthalene intoxication in sheep.

The post -mortem findings were necrosis and cirrhosis of the liver. Watson and Hunter ( 1958) studied the pathology of Lesteria infection in lambs and described that liver revealed multiple white necrotic foci uniformely distributed throughout the liver substance. Lesions varied from 1-7 m.m. in diameter. Baxter ( 1959) observed the histopathology of aldrin poisoing in lambs and described that the liver lesion was of an acute toxic hepatitis in which a well marke central necrosis had under gone repair by regeneration of liver cellsx and to a minor degree by replacement with fibrous tissue. Quesada et al ( 1960) gave an account of necrotic hepatitis caused by Fusiformis necrophorus in lambs aged 15-20 days. Muth(1960) studied the pathology of carbon tetrachloride poisoning of ewes on low selenium diet. They described that the charactarstic lesion was hepatic centrilobular haemorrhagic necrosis. Gallaghar (1962) studied the effect of drenching technique on poisoning of sheep with carbon tetrachloride and described that those killed by administration of the mixture showed severe congestion and diffuse haemorrhagic centrilobular necrosis of the liver. Nagy ( 1963) studied the pathology of Pasteurella pseudotuberculosis infection in sheep and described that post martem examination revealed numerous necrotic foci in the substance of the liver, kidney, spleen and lungs. Histologically they consisted necrotic material surrounded by cellular infilteration and bacteria. Setchell and Little Johnes ( 1963) described the liver histopathology

of sheep with anthelmintic dose of carbon tetrachloride. The liver sections examined showed in most cases either centrilobular necrosis, fatty degeration, cloudy swelling or no abnormality . Jubb and Kennedy ( 1963) described the pathology of massive hepatic necrosis in sheep and reported that it is probably nutritional but it was not confirmed. They further reported that it is frequently associated with pastures in which birdsfoot trefoil ( Lotus tenuis) is abundant . Koppang et al ( 1964) studied the pathology of feeding experiments with meal produced from herring preserved with sodium nitrite and formaline. They described that acute cases were characterised by extensive liver necrosis, mainly centrilobular. In chronic cases, the main findings were a non portal fibrosis with obliterating changes inthe central and sublobular vein.

## R E S U L T

Gross pathology:-

The liver was pale in patches. In one of the specimen there was an irregular area of caseation necrosis . The area around the caseation was severely congested. On incision the area of caseation followed deep into the liver substance.

Histopathology:-

Microscopically there were areas of diffuse

of sheep with anthelmintic dose of carbon tetrachloride. The liver sections examined showed in most cases either centrilobular necrosis, fatty degeration, cloudy swelling or no abnormality . Jubb and Kennedy ( 1963) described the pathology of massive hepatic necrosis in sheep and reported that it is probably nutritional but it was not confirmed. They further reported that it is frequently associated with pastures in which birdsfoot trefoil ( Lotus tenuis) is abundant . Koppang et al ( 1964) studied the pathology of feeding experiments with meal produced from herring preserved with sodium nitrite and formaline. They described that acute cases were characterised by extensive liver necrosis, mainly centrilobular. In chronic cases, the main findings were a non portal fibrosis with obliterating changes inthe central and sublobular vein.

## R E S U L T

Gross pathology:-

The liver was pale in patches. In one of the specimen there was an irregular area of caseation nmcrosis . The area around the caseation was severely congested. On incision the area of caseation followed deep into the liver substance.

Histopathology:-

Microscopically there were areas of diffuse

coagulative necrosis . The cellular architecture was present where as the cellular details were absent. At the junction of the normal tissue and necrosed tissue there were infilteration with leucocytes comprising neutrophils and mononuclear cells ( Fig. 30) . The specimen showing caseation microscopically revealed homogenous area taking an intense eosinophilic stain. Neither cellular architecture nor the cellular details were present, giving the appearance of caseous necrosis ( Fig. 31) . The area adjacent to caseous necrosis were showing coagulative necrosis surrounded by leucocytes. The area of necrosis was engorged with erythrocytes. The lesions were suggestive of haemorrhagic caseonecrotic hepatitis.

## D I S C U S S I O N

Various etiological factors have been incriminated by various authors in causation of the hepatic necrosis. Runnels et al ( 1965) have reported that variety of bacteria, parasites, chemical and plant poisons are the cause of hepatic necrosis. In addition Jubb and Kennedy ( 1963) have reported that probably nutritional factors are also responsible for necrosis but it was unconfirmed. In the present investigation no bacteria or parasites were seen on microscopical examination. The necrosis may be due to some chemical or plant poisons or bacteria which might have been few in number and escaped detection under microscope.

## ABCESS in LIVER

## I N T R O D U C T I O N

Abcess formation is an important entity under suppurative inflamation of tissues. Abcess formation is one of the defence mechanisum. Hepatic abcesses are encountered in almost all domesticated animals.

## REVIEW OF LITERATURE

Dickinson ( 1962) observed hepatic abcesses in calf. The liver weighed 12.5 lbs. and was studded with raised nodules having purulent centre. Corynebacterium pyogenes was isolated from those lesions. Newsom ( 1938) reported the occurrence of the liver abcess and pointed out that the liver abcess in cattle of Colorado was mainly due to the feeding of beef by-products to these animals. Smith ( 1944) pointed out that rumen ulcers are a source of abcess formation in the liver, which was supported by Jensen et al ( 1954). Latter workers observed that hepatic abcess was more common in cattle with lesions in the rumen. They also isolated F.necrophorus organisms from the anterior ventral sac of rumen. Jensen et al ( 1947) demonstrated liver abcess in cattle after experimental feeding of cattle on rich concenterate diet with low fibre content. Robinson ( 1951) reported the natural occurrence of liver abcess in cattle. Experimentally they were unable to

reproduce liver abcess or any type of liver damage after drastic change in the feed and even after oral feeding and intraveinous inoculation of culture of F.necrophorumx .

Jensen et al (1954) experimentally produced abcess in 33 cattle after inoculation of F. necrophorus in the portal system. They found on autopsy that after a day of inoculation the abcess were encountered in the liver parenchyma. They found that upto 6th day of of inoculation the lesions were characterised by coagulative necrosis with leucocytic infilteration and colonies of bacteria were seen. At the periphery of the lesions there were few neutrophils . The area surrounding the lesions showed pyknotic and chromatolytic nuclei to-gether with accumulation of mononuclear cells in the sinusoids. After 8 -10 days the chief lesions were coagulative necrosis, formation of pus and encapsulation. In the centre of the lesions there were xix polymorphs and macrophages . The encapsulation was noticed to comence from the 6th day of inoculation. The inner layer of capsule consisted of immature fibroblasts, capillaries with an abundance of lymphocytes , macrophages and plasma cells with a few polymorphs . The outer layer was in direct contact with the parenchyma . Replacement of abcesses by fibruus tissue scar was at stages varying from 45-180 days after inoculation.

Gaiger and Davies ( 1957) described that pus is produced by the destruction and lequifaction of tissue

cells and exudation of lymph. They contain chiefly neutrohils of which many are degenerated and necrotic. They may also contain organisms. They further described that they are surrounded by wall of granulation tissue, the pyogenic membrane, Kater et al ( 1962) described in a case of specific sup urative synovitis and pyaemia in lambs that abcesses were always present in the liver. Romboli et al ( 1963) described that liver abcesses are common in calves and lambs with omphalophlebitis.

## R E S U L T

Gross Pathology:-

There was yellowish white focus measuring about ½ inch in diameter on the surface. When it was incised a cavity containing caseated mass was encountered into the substance of the liver.

Histopathology:-

The lesion consisted of inspissated pus surrounded by a fibrous tissue capsule. The inspissated pus consisted of degenerated neutrophils and few round cells, together with fused fibrin. The inner layer of connective tissue capsule consisted of immature fibroblasts, abundance of round cells, plasma cells and few neutrophils. The outer layer of the connective tissue capsule was in direct contact with the parenchyma. In addition, the section also

revealed proliferation of newly formed bile ducts. The wall of the bile duct was thickened. Proliferation of fibrous tissue around the bile ducts and infilteration of the portal triads by round cells were also evidenced.

## DISCUSSION

Various etiological factors have been incriminated in the causation of the hepatic abcesses. Flynn( 1946) reported that the pyogenic abcesses in human being is caused by Colon bacilli, Streptococci and Staphylococci. Hepatic abcesses in cattle is caused by F. necrophorus (Newsom (1938) Jensen et al(1954) Dickinson(1932 ). Newsom (1938) reported Corynebacterium pyogenes responsible for hepatic abcesses.

In the present investigation although isolation was not adopted, the sections stained by Gram's method revealed Gram positive, thin short rods morphologically indistinguishable from diphtheroids, which might be the causative agent.

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## EOSINOPHILIC HEPATITIS

## R E S U L T

Gross Pathology:-

The liver was congested. On the surface of the liver there was a greyish white focus measuring about 3-4 m.m. in diameter.

Histo pathology:-

The central vein and sinusoids were engorged with erythrocytes. The central vein was dixlated. Hepatic cells were showing vacuolization and individualization. The hepatic cells were showing various degrees of degenerative changes and necrosis at places. Kupffer cells were prominent. In a portion of section there were a infilteration of mononuclear leucocytes. The section also revealed heavy infilteration of eosinophils. There were whorl formation indicating tendency of giant cell formation. In the area of infilteration the hepatic cells were completely replaced by eosinophilsx and at certain places only islands of few atrophied and degenerated hepatic cells were seen (Fig.32,33)

## DISCUSSION

Available literature does not describe about xxs this type of eosinophilic infilteration in liver of goats.

So this might be a case of eosinophilic hepatitis. However mononuclear cell infilteration and tendency of giant cellx formation are indicative of chronic hepatitis.

Zendulka, ( 1960) has described about chronic interstial eosinophilic hepatitis in pigs due to migrati n of Ascaris larvae. In the present investigation the gross lesions were not suggestive of Ascaris larvae migration. More over cut section of larvae were also not seen in the section.

However the etiology for this type of lesion could not be established. As such this aspect needs further experimental elucidation.

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## TUBERCLE LIKE NODULES

## R E S U L T

**Gross Pathology:-**

The liver was congested. There were white caseous nodules of mustard seed size scattered through out the surface of the liver. On incision these nodules were also present in the substance of the liver.

**Histopathology:-**

There were central necrosed area surrounded by fibrous tissue containing different types of leucocytes mainly round cells and occasional plasma cells (Fig. 34) . At places there were areas of coagulative necrosis surrounded by leucocytes. At one place the central vein was immensly dilated and its wall was very thick. Portal triads were showing infilteration of round cells which was slightly more than normal . The portal triads were also showing proliferation of newly formed bile ducts. Hepatic cells were showing various degrees of degenerative changes . One of the specimen was showing engorgement of central vein and sinusoids with erythrocytes together with dilatation of the central vein .

## DISCUSSION.

Available literature does not describe this type of lesions. Moreover neither any bacteria nor any parasite could be found in the sections. As such the etiology for this type of lesion could not be established. However this aspect needs further experimental elucidation.

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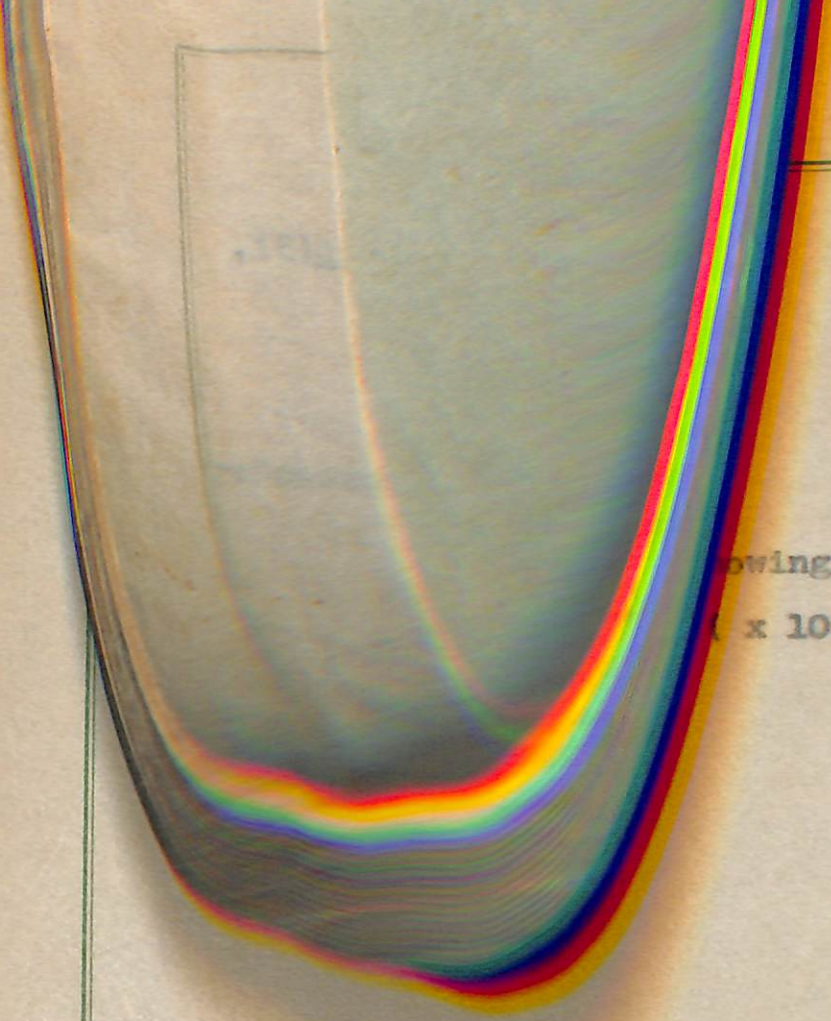
# PHOTOGRAPHS

Fig. 1- Liver showing few haemorrhagic spots on the surface.

Fig. 2- Common bile duct showing nodules on the mucosa and attached Amphistomes.

Fig. 3- Section of liver showing infiltration with leucocytes . ( x 100)

Fig. 4- Section of liver showing fibrosis of the portal triad, dilatation of portal vein, thickening of the arterial wall and biliary hyperplasia .( x 100 )



owing infiltration

x 100)

Fig. 4- Section of liver showing fibrosis of the portal triad, dilatation of portal vein, thickening of the arterial wall and biliary hyperplasia .( x 100 )

Fig. 5- Section of common bile duct showing Amphistome attached with a flask shaped projection of the mucosa. (x 25)

Fig.6- Section of Common bile duct showing hypertrophy, hyperplasia , leucocytic infiltration, necrosis and desquamation of the epithelium . ( x 100)

Fig. 7- Section of common bile duct showing fibrosis of the outer layer and infiltration with leucocytes . ( x 100)

Fig.8- Section of Common bile duct showing fibrosis, atrophy of the smooth muscle and thickening of the arterial wall.(x100)

Fig. 9- Liver showing haemorrhagic spots on the surface.

Fig. 10- Liver showing irregular elevations and depressions due to atrophy and fibrosis.

Fig. 13- Section of liver showing haemorrhagic spots and tracts . ( x 100)

Fig. 14- Section of liver showing haemorrhagic tract containing cut section of Fasciola amidst the exudate containing erythrocytes and leucocytes. ( x 100)

Fig. 15- Higher magnification of Fig.14 showing cuticular spines of the Fasciola and different leucocytes and erythrocytes present arou nd the parasite . ( x 400 )

Fig. 16- Section of liver showing (i) healed up haemorrhagic tracts as scars infilterated with leucocytes (ii) andx irregular fibrosis due to fusion of scars. ( x 100)

Fig.19- Section of bile duct showing proliferation, desquamation and leucocytic infileration in fascioliasis. ( x 100 )

Fig. 20- Section of liver showing cut sections of the eggs of Fasciola within the bile duct .( x 100)

Fig. 21- Liver showing Hydatid cysts.

Fig. 22- Section of liver showing brood capsules containing scolices attached to the germinal layer surrounded by a thick outer concentrically laminated connective tissue capsule ( x 100)

Fig. 23- Section of liver showing infilteration with mononuclear cells at the junction of hepatic parenchyma and fibrous tissue capsule of thea cyst. ( x 400)

Fig.24- Section of liver showing pericellular cirrhosis in Hydatidiosis (x 100)

Fig.25- Section of liver showing monolobular cirrhosis in Hydatidiosis . ( x100)

Fig. 26- Section of liver showing disorganisation and degeneration of cyst wall in advance stage of Hydatidiosis .( x 100)

Fig. 27- Section of liver showing infiltration with mononuclear cells interspersed with few giant cells around the cystic structure in advance stage of Hydatidiosis ( x 900)

Fig. 28- Liver showing mottled appearance and severe congestion.

Fig. 27- Section of liver showing infiltration with mononuclear cells interspersed with few giant cells around the cystic structure in advance stage of Hydatidiosis ( x 900)

Fig. 28- Liver showing mottled appearance and severe congestion.

Fig. 29- Section of liver showing engorgement of sinusoids with erythrocytes and fatty changes. ( x 400)

Fig. 30- Section of liver showing coagulative necrosis with leucocytic infiltration. ( x 100)

Fig. 31- Section of liver showing caseation necrosis, the architectural and cellular details are lacking. The area is surrounded by leucocytes. ( x 100)

Fig. 32- Section of liver showing infilteration with eosinophils ( x 400)

Fig.33- Higher magnification of
Fig. 32. ( x 900)

Fig. 34- Section of Tubercle like nodule from liver showing central area of necrosis surrounded by fibrous tissue capsule infiltrated with leucocytes. ( x 100)

# C L A S S I F I C A T I O N

# C L A S S I F I C A T I O N

According to Himsworth ( 1954) liver deseased are classified as follows:-

1. Infilteration - (a) Intrinsic
   (b) Extrinsic
2. Parenchymatous Hepatitis-(a) Acute (i) Zonal
   (ii) Massive.
   (b) Subacute- Massive
   (c) Chronic (i) Post necrotic scarring(Chronic massive hepatitis)
   (ii) diffuse hepatic fibrosis.
3. Biliary lesions (a) Obstructive
   (b) Cholangio -hepatitis.
4. Circulatory lesions (a) Absolute ischaemia
   (b) Relative ischaemia
5. Focal lesions - (a) Inflamatory
   (b) Cysts
6. New growthsxx - (a) Primary
   (b) Secondary.

Smith and Jones ( 1958) have classified

hepatitis as Infectious hepatitis and Non infectious hepatitis . Non Infectious hepatitis has been further classified as Acute toxic hepatitis and Chronic toxic hepatitis.

In the present studies it is not possible to classify liver diseases on the basis of etiology because in many cases the etiology chould not be established, as such the liver diseases that have been encountered have been classified on the basis of pathological changes as follows:-

1. Parenchymatous hepatitis

(a) Acute-0.274% of cases were showing changes of Acute parenchymatous hepatitis. The lesions were characterised by massive necrosis in three cases, abcess formation in one case and individualization of hepatic cells accompanied by icterus in three cases.

(b) Chronic :- 6.409% of cases were showing changes of chronic parenchymatous hepatitis characterised by haemorrhage, hyperaemia ,exudative changed and proliferation of fibrous tissue.

2. Biliary lesions:- 3.22% of cases were showing biliary lesions characterised by hyperplasia of the epithelial lining, proliferation of fibrous tissue, exudative

changes accompanied by necrosis and desquamation.

3. Circulatory lesions:- 2.78% of cases were showing circulatory lesions characterised by congestion.

During the present studies one unusual case of Eosinophilic hepatitis characterised by necrotic changes , various degenerative changes and massive eosinophilic infiltration and two unusual cases of tubercle like nodules characterised by cen ral area of necrosis surrounded by fibrous tissue infilterated with lymphoid cells were encountered.

S U M M A R Y

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# S U M M A R Y

# S U M M A R Y

In the present investigation the specimens were collected from 244 goats showing a pathological condition encountered, when 2,546 goats were surveyed and examined for the incidence.

1. One case of Biliary Amphistomiasis was recorded. On examination the parasite was identified as Gigantocotyle explanatum . Pathological lesions were suggestive of transperitoneal root of migration of the parasite. The lesions were characterised by haemorrhagic tracts, fibrosis, biliary hyperplasia in the liver , together with fibrosis , hyperplasia and other inflamatory changes in the larger bile ducts.

2. 82 cases of Fascioliasis was recorded. On examination the parasite was identified as Fasciola gigantica. Pathological lesions were suggestive of transperitoneal root of migration of the parasite. The lesions were characterised by haemorrhagic tracts, fibrosis, biliary hyperplasia , mucus degeneration of biliary epithelium.

3. 80 cases of Hydatidiosis was recorded. In early stage the lesions were characterised by fibrosis, various degenerative changes and necrosis, in advance stage

the lesions were characterised by degeneration of parasitic cyst wall, fibrosis, presence of giant cells around the cyst wall.

4. Congested livers were mostly seen in goats. The lesions were characterised by engorgement of central vein and sinusoids with erythrocytes accompanied by fatty changes and mild fibrosis.

5. 3 cases of icterus was recorded and the lesions were characterised by disorganization of hepatic cords and individualization of hepatic cells which is an indication of onset of necrosis.

6. Two cases of coagulative necrosis and one case of caseous necrosis was also recorded.

7. One case of hepatic abcess was recorded. Small thin Gram postive rods morphologically indistinguishable from diphtheroids were seen in the section, which might be the causative agent.

8. One case of Eosinophilic hepatitis characterised by unusual accumulation of eosinophils together with lymphoid cells was recorded.

9. In two cases tubercle like nodules were recorded. The lesion was characterised by central necrosed area

surrounded by fibrous tissue infilterated with mononuclear leucocytes .

Liver diseases encountered have also been classified on the basis of pathological lesions.

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