Pathological Studies on Liver affections in Saudi Arabia Camels

By

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ABSTRACT

In the present investigation, 500 liver of One humped slaughtered camel at abattoirs in Saudi Arabia were examined. Out of these examined liver, 75 livers showed different hepatic lesions with an incidence of 15%. According to the microscopic findings, these hepatic lesions were classified into acute hepatitis observed in three cases, suppurative hepatitis in four cases, six cases of parasitic hepatitis were diagnosed. granulomatous hepatitis observed in two cases. Different types of hepatic cirrhosis were diagnosed in 24 cases of the total hepatic lesions, the hepatic cirrhosis represented by portal cirrhosis, biliary cirrhosis, central cirrhosis and glissonian cirrhosis, degenerative changes in the form of fatty, hydropic and vacuolar degenerations were recorded in 29 cases, chronic venous congestion in three liver, two hepatic neoplasms including metastatic lymphosarcoma and hemangioma together with osteolipomatous metaplasia were also observed.

INTRODUCTION

One humped camels (Camelus dromedarius) are very important in many Arabian and gulf countries, as they are used as food and draft animals, and have also been tapped as an important sport and tourism resource in the Arabian Gulf countries. The ability of camels to utilize range in marginal areas and to
survive and produce under harsh environmental conditions has been recognized over the years (Hjort and Hussein 1986, Abbas and Tilley 1990 and Schwartz 1992).

The liver of dromedary camel contains a high amount of interlobular connective tissue leading to a firmer consistency than in other domesticated animals (Abdalla et al. 1971). Liver diseases are relatively common but often occurs in the absence of specific clinical signs. The liver has considerable power of regeneration, severe loss of hepatic cells can be restored and normal architecture is retained. However, if the loss of the hepatic cells is more severe cirrhosis can result. Cirrhosis is an irreversible disturbance which may affects the entire liver and may regenerate in a nodular pattern. The regenerating nodules lack the organized lobular structure of the normal liver and the blood haphazardly reach to it resulting in an inefficient organ that is prone to liver failure. Liver failures do not appear until 70-80% of the functional capacity is lost.

Hydatid liver disease and migration of the immature flukes especially Fasciola hepatica, produces hemorrhagic tracks of necrotic liver parenchyma (Elbihari 1985; Fahmy and El_Attar, 1990 and Zukowski et al. 1992). Mature flukes reside in the larger bile ducts and cause cholangitis or cholangiohepatitis which may lead to stenosis of the duct (Fahmy and El_Attar, 1990; Behm and Sangster 1999 and Eslami et al., 2003).

Different types of degenerative changes and liver diseases of camels such as hepatic lipidosis with biliary hyperplasia, hepatic necrosis, cholangiohepatitis, cholangitis, pericholangitis, and septic phlebitis were studied and reported by (Sakr et al., 1991, Tej Singh et al. 2006 and Anderson 1999).

Hepatic affections of camels usually associated with different
causative agents which including bacteria, virus, parasites and fungi (Mohamed, et al., 1997).

The present study was designed to investigate the common hepatic lesions in liver of camelus dromedaries slaughtered in abattoirs of the different localities in Saudi Arabian Kingdom with full description of the gross and microscopic findings of each lesion.

MATERIALS AND METHODS

A total of 500 liver of camels of different sexes and ages slaughtered at abattoirs in different localities in Saudi Arabia Kingdom during the period from 2008 to 2010 were examined. The gross appearance, location and size of the lesions were recorded. Among these examined liver, 75 showed different hepatic lesions in a percentage of 15% of the total examined cases.

Out of these examined liver, small tissue specimens were collected from seventy five liver showed different hepatic gross lesions. These tissue specimens were fixed in 10% neutral buffer formalin. After proper fixation, specimens were washed under tap water, then dehydrated in ascending strength of ethyl alcohol, cleared in xylol and embedded in paraffin. Five microns paraffin sections were prepared and stained with hematoxylin and eosin for general microscopic examination (Bancroft et al., 1994).

Moreover, special staining techniques such as Masson's trichrome stain, Von Kossa stain, and PAS stain were also carried out for demonstration of connective tissues, calcium salts, and parasitic elements respectively (Durury and Wallington, 1984)

RESULTS & DISCUSSION

The present study was carried out to determine the most common hepatic lesions in camels slaughtered at abattoirs in Saudi Arabia and out of
500 examined liver of slaughtered camels, 75 showed different hepatic lesions with an incidence of 15%. According to the microscopic findings, these hepatic lesions were illustrated in table (1).

**Table (1)** Types, number and percentage of the recorded hepatic lesions.

<table>
<thead>
<tr>
<th>Types of hepatic lesion</th>
<th>Number of affected cases</th>
<th>% of affected cases from total No of examined liver</th>
<th>% of affected cases from total hepatic lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1- Toxic hepatitis</td>
<td>3</td>
<td>0.6</td>
<td>4.00</td>
</tr>
<tr>
<td>2- Suppurative hepatitis</td>
<td>4</td>
<td>0.8</td>
<td>5.33</td>
</tr>
<tr>
<td>3- Parasitic hepatitis</td>
<td>6</td>
<td>1.2</td>
<td>8.00</td>
</tr>
<tr>
<td>4- Granulomatous hepatitis</td>
<td>2</td>
<td>0.4</td>
<td>2.67</td>
</tr>
<tr>
<td>5- Hepatic cirrhosis</td>
<td>24</td>
<td>4.8</td>
<td>32.00</td>
</tr>
<tr>
<td>Portal cirrhosis</td>
<td>6</td>
<td>1.2</td>
<td>8.00</td>
</tr>
<tr>
<td>Biliary cirrhosis</td>
<td>8</td>
<td>1.6</td>
<td>10.67</td>
</tr>
<tr>
<td>Central cirrhosis</td>
<td>6</td>
<td>1.2</td>
<td>8.00</td>
</tr>
<tr>
<td>Glissonian cirrhosis</td>
<td>4</td>
<td>0.8</td>
<td>5.33</td>
</tr>
<tr>
<td>6- Degenerative changes</td>
<td>29</td>
<td>5.8</td>
<td>38.67</td>
</tr>
<tr>
<td>Fatty degeneration</td>
<td>15</td>
<td>3</td>
<td>20.00</td>
</tr>
<tr>
<td>Hydropic degeneration</td>
<td>14</td>
<td>2.8</td>
<td>18.67</td>
</tr>
<tr>
<td>7- Chronic venous congestion</td>
<td>3</td>
<td>0.6</td>
<td>4.00</td>
</tr>
<tr>
<td>8- Hepatic neoplasm</td>
<td>2</td>
<td>0.4</td>
<td>2.67</td>
</tr>
<tr>
<td>□ Metastatic lymphosarcoma</td>
<td>1</td>
<td>0.2</td>
<td>1.33</td>
</tr>
<tr>
<td>□ Hemangioma</td>
<td>1</td>
<td>0.2</td>
<td>1.33</td>
</tr>
<tr>
<td>9- Osseous metaplasia</td>
<td>2</td>
<td>0.4</td>
<td>2.67</td>
</tr>
<tr>
<td>Total</td>
<td>75</td>
<td>15</td>
<td>100</td>
</tr>
</tbody>
</table>

Regarding with toxic hepatitis, three cases were detected in this work (4% of the affected cases). Macroscopically, the liver was swollen, red, and blood oozed on cut section. Irregular pale yellowish areas on the hepatic surfaces were seen. This result was partially agreed with Mohamed, et al., (1997) where similar gross pictures with the presence of grayish foci on hepatic surface were seen in liver of camel.
suffered from Clostridium infection.

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Microscopically, severe congestion and diffuse hemorrhages with hemosiderosis were seen. In addition, thrombosis of the portal vessels with severe fatty change and focal and confluent areas of hepatic necrosis were detected. Lymphocytic infiltration with biliary hyperplasia was also noticed. Disruption of the lobular architecture with inflammatory cells in the sinusoids (Fig.1) with pericentral necrosis replaced by mononuclear cells especially macrophages (Fig.2). Ground glass cells containing homogenous eosinophilic deposits (individual cells necrosis) with variations of nuclear size and presence of binucleated cells was observed (Fig.3). These microscopic pictures were similar to those mentioned by Abd El-Samee (1998) in acute hepatitis, Mohamed, et al., (1997) in liver of camel suffered from Clostridium infection, Abu Damir, et al., (1989) and Nawal Osman et al. (2004) in liver of camel in cases of experimental copper poisoning.

Suppurative hepatitis was recorded in 5.33% of the affected cases. This incidence disagreed with that reported by Al-Ani, et al. (1998) who added that, liver abscesses were diagnosed in 1.2% of the liver of slaughtered camel.

Grossly, the liver was congested and showed multiple small foci contained whitish caseated pus. Microscopically, three cases of
chronic suppurative hepatitis evidenced by focal area of necrosis enclosed by thick capsule and inflammatory reaction were diagnosed (Fig.4). In one case, areas of liquifactive necrosis enclosed by neutrophils and mononuclear inflammatory cells with hydropic degeneration of hepatic cells in vicinity of abscesses were noticed. Similar macroscopic and microscopic pictures were also mentioned by Mohamed, et al., (1997) in the liver of slaughtered camel due to Streptococcus and Corynebacterium infections, in Sharkia province, Egypt, and Itman et al. (1989) in the liver of camel in case of Cl. Perfrinengs and Cl. sordellii infections.

Six cases of parasitic hepatitis (8%) were diagnosed in this work. While Fouad (2002) mentioned that; the incidence of parasitic hepatitis in camel was 16.34 %, in addition Eslami, et al. (2003) reported that 5.3% of the examined liver of camel harbored F. hepatica, In parasitic hepatitis the liver was greatly firm, pale and showed grayish white foci with thickened hepatic capsule. In some cases, the wall of bile ducts was thickened and whitish in color. These findings were supported by Fahmy and El-Attar (1990) and Fahmy et al. (1998) who described similar gross lesions in the liver of camel infested by fascioliasis.

Microscopically, numerous migrating tracks contained sections of parasites surrounded by inflammatory cells mainly eosinophils were noticed in some cases (Fig.5), while in other cases these tracks were formed from central necrotic hepatocytes surrounded by inflammatory reaction mainly eosinophils and mononuclear cells. These microscopic changes were come in harmony with those reported by Eslami, et al. (2003). Hepatic degeneration and necrosis with mononuclear cells aggregation were also found. Extensive fibrosis, bile ducts hyperplasia with periductal mononuclear cells aggregation and
fibrosis were also reported (Fig.6). These results were agreed with that recorded by Fahmy and El-Attar (1990), Wahba, et al. (1997) and Fahmy et al. (1998). A number of examined liver, lesion of parasitic cirrhosis was observed manifested by replacing of most of the hepatic parenchyma by fibrous connective tissue and the portal tracts contained proliferating fibrous connective tissue and the bile ducts were dilated and lined by flattened epithelium (Cystic adenomatous appearance) with the presence of brownish masses inside the lumens of some of these ducts (Fig. 7).

In the present investigation, two cases of granulomatous hepatitis were diagnosed (2.67%) in addition to some cases in relation with other lesions especially hepatic cirrhosis. Microscopically, multiple granulomas formed either from aggregation of mononuclear cells enclosed by connective tissue capsule or focal hepatic necrosis enclosed with mononuclear inflammatory cells and multinuclteated giant cells (Fig.8). Other granulomas consisted of large caseated area with calcification enclosed by mononuclear cells, epithelioid cells, few giant cells and enclosed by thick fibrous tissue capsule, meanwhile other granulomas formed from large caseated area surrounded by fibrous connective tissus containing mononuclear cells.

In case of biliary cirrhosis some granulomas consisted of severe destruction of the bile duct and surrounded by large number of mononuclear cells and fibrous tissue (Fig.9), Near to these granulomas, the hepatic cells were suffered from vacuolar and hydropic degeneration. These microscopic pictures were similar to those mentioned by Sakr, et al.,(1991) in the liver of Egyptian camels suffered from trypanosome infection.

Moreover other granulomas were also observed especially in case of biliary cirrhosis consisted of large
caseated area enclosed by fibrous tissue and mononuclear cells and some others, consisted of severe destruction and necrosis of the bile duct surrounded by large number of mononuclear cells and enclosed by fibrous tissue.

Different types of liver cirrhosis were diagnosed and represented the main hepatic lesions (32% from the total hepatic lesions). Depending on the size of the nodules there were three macroscopic types: micronodular, macronodular and mixed cirrhosis. In micronodular form (portal cirrhosis) regenerating nodules were under 3 mm. In macronodular cirrhosis (post-necrotic cirrhosis).

Concerning nodular (portal cirrhosis) it was seen in six cases (8%). Macroscopically; the liver was firm, grayish white with thickened interlobular septa and marked lobulation. Microscopically; The microscopic examination of the liver revealed nodular formation of the hepatic lobules consisted of fibrous connective tissue proliferation enclosed regenerated hepatic cells with absence of central veins (Fig.10), crossman stain revealed fibrous connective tissue proliferation infiltrated by mononuclear cells and enclosed islands of few atrophied hepatic cells with the absence of central veins and contained numerous blood capillaries and nonfunctional bile ducts and ductules extending between portal regions was found (Fig.11). Fibrous expansion form bridging necrosis and fibrosis from the portal areas to other portal areas Porto-portal and between portal area and central veins Porto-central were also seen (Fig.12). The portal tracts enlarged and infiltrated by chronic inflammatory cells with hyperplastic bile ducts and periductal mononuclear cells infiltration and fibrosis (Fig.13). In some cases thrombus was observed in the portal vein (Fig.14).

Eight cases of biliary cirrhosis were noticed (10.76%). Macroscopically, the liver was firm,
grayish brown and on cut sections revealed thickening of the bile duct wall. Microscopically, bile duct hyperplasia and/or severe destruction and desquamation of its lining epithelium with peribiliary fibrosis and mononuclear aggregation with formation of newly formed bile ductules were noticed. In some cases, bile ducts were severely impacted by cell debris and inflammatory cells with biliary hyperplasia associated with peribiliary mononuclear cellular aggregation and formation of newly formed non functional bile ductules (Fig.15). Moreover, some bile ducts showed severe necrosis and sloughing of its lining epithelium and markedly surrounded by fibrous connective tissue. The hepatic cells were suffered from degenerative changes in the form of hydropic and vacuolar degeneration together with fatty change (Fig.16). Bile pigments were accumulated in the bile canaliculi forming bile thrombi with atrophy of the hepatic cells (Fig. 17), bile was also accumulated in the hepatic lobules of the liver (cholestasis) due to severe injury and loss of hepatocytes to form lakes of bile (Fig.18). Micronodular cirrhosis was prominent which was formed from loss of hepatocytes with replacement fibrosis that enclosing the regenerated nodules (Fig.19).

These microscopic pictures were also described by Fahmy, et al (1993) and Gameel et al. (1994). Moreover, the hepatic cells were suffered from hydropic and vacuolar degeneration. Rubbins et al., (2007) added that the portal tracts were infiltrated by lymphocytes, macrophages, plasma cells, and few eosinophilis. Our opinion is that the intrahepatic bile duct obstruction leads to upstream bile ductular proliferation, inflammation and necrosis of the adjacent periportal hepatic parenchyma, with generalized cholestasis, portal tract scarring and bridging fibrosis which finally leads to biliary cirrhosis.
Six cases of central cirrhosis were diagnosed (8%). Microscopically, fibrous connective tissue proliferation replaced the hepatic cells around central veins with degeneration of the adjacent hepatic cells were found (Fig. 20). In some cases fibrous bridges were extended from the pericentral fibrous tissue toward the hepatic lobules (Fig.21&22)

Concerning Glissonian cirrhosis Four cases were reported (5.33%). Grossly, liver was brownish gray, firm with thickened hepatic capsule. Microscopically, marked thickening of the hepatic capsule due to fibrous connective tissue proliferation with hydropic and vacuolar degeneration of the adjacent hepatocytes were recorded (Fig.23,24). These findings were nearly similar to that described by Egbe Nwiyi and Chaudhry (1994) who observed the same results in addition to mononuclear inflammatory cells infiltration of the portal areas was also seen.

In the present investigation, 29 cases (38.67%) showed various types of degenerative changes. These hepatic degenerative changes were represented mostly by fatty changes and hydropic degeneration in addition; two cases showed amyloid infiltration in combination with hydropic degeneration. This result was supported by Tej Singh et al. (2006). Microscopically, focal or diffuse fatty degeneration of hepatocytes with mononuclear cells infiltration in the hepatic parenchyma and portal areas was seen.

Concerning hydropic and vacuolar degeneration, the liver was slightly soft with an irregular pale brown areas. Microscopically, hydropic degeneration appeared either focal or diffuse. Moreover, focal aggregation of lymphocytes in-between hepatic cells was also observed. Sometimes the affected hepatocytes were markedly swollen and contained ill defined pale eosinophilic vacuoles in their cytoplasm with normal or slightly pyknotic nuclei.
Three cases of CVC of the liver were diagnosed with an incidence of 4% of the affected cases. Microscopically, severe congestion and central cirrhosis with centrolobular hepatic necrosis were found. Moreover, hydropic and fatty degeneration of the hepatic cells was seen at the periphery of hepatic lobules.

Two cases of hepatic neoplasms were recorded (2.67%). These neoplasms were in the form of cavernous hemangioma in one case and other case was diagnosed as metastatic lymphosarcoma.

Cavernous hemangioma, appeared grossly as small round dark red spongy elevated mass filled with blood. Microscopically, it was formed from irregular spaces filled with blood and lined with single layer of endothelial cells (Fig. 25). The adjacent hepatic cells were suffered from necrosis and pressure atrophy. similar findings were described by El-Mahdy et al. (1997) in cavernous hemangioma in the liver of camels.

Concerning metastatic lymphosarcoma, it was appeared as multiple grayish white circumscribed masses embedded in the hepatic parenchyma. The cut section of such masses was grayish white and separated from the surrounding hepatic parenchyma by thick whitish capsule. Microscopically it was represented by aggregated pleomorphic neoplastic cells contained pleomorphic vesicular nuclei with prominent nucleoli and mitotic activities. These neoplastic masses were surrounded by thick capsules and inflammatory cellular reaction (Fig. 26). This microscopic picture was in accordance with those of Simmons, et al., (2005) in the liver of camel in multicentric T-cell lymphoma.

Cholangiocarcinoma was observed in two cases of biliary cirrhosis, consisted of clearly defined well differentiated glandular and
tubular structures lined by anaplastic cuboidal to low columnar epithelial cells, the nuclei were round to oval, vesicular and hyperchromatic with variable degrees of mitotic activities (Fig.27). It may form glandular structures of varying size and shapes, with hyperchromatic round or oval nuclei and surrounded by dense fibrous stroma (Fig.28&29). These findings were in close similarity to those mentioned by Jones and Hunt (1979), Mc-Gavin and Zachary (2007) and Shinohara, (2009). Cholangiocarcinoma was arisen from the intrahepatic or extrahepatic biliary epithelium (Darwin, et al., 2009 and Shinohara, 2009). They added that cholangiocarcinoma was frequently associated with conditions that cause chronic liver inflammation and cirrhosis such as liver fluke infestation, sclerosing cholangitis, and chronic viral hepatitis.

In the present investigation, two cases of osteolipomatous metaplasia were recorded (0.2%). Macroscopically, irregular solid nodules were embedded in the liver. The cut sections of these masses showed hard whitish contents with gritty sound. Histopathologically, these nodules consisted of an aggregation of metaplastic osseous plates formed from osteoblasts and clusters of large vacuolated cells resembling fat cells with areas of calcification (Fig.30). These results were in agreement with those reported by Al-Sadi, (1994) and El-Mahdy, et al. (1997), the hepatic cells adjacent to these osseous plates were suffered from degenerative changes and pressure atrophy. Thickening of the interlobular septa and peribiliary fibrosis were also found.

Concerning other lesions associated with the above mentioned findings, peliosis hepatis characterized by multiple dilated blood filled cysts without distinct wall. In some cases the large blood cysts scattered throughout the liver, and the wall consisting of an
uninterrupted thin layer of reticulin fibers (Fig.31), while telangiectases which occur throughout the liver as dark red areas, irregular in shape but well circumscribed, and ranging from pinpoints to many centimeters in size, characterized microscopically by marked dilatation of thin walled blood filled sinusoids (Fig.32).
Figure (1) Toxic hepatitis: Disruption of the lobular architecture with mononuclear cells in the sinusoids. (Stain H&E, X 400).

Figure (2) Toxic hepatitis: pericentral necrosis replaced by mononuclear cells especially macrophages. (Stain H&E, X 400).

Figure (3) Toxic hepatitis: Ground glass cells containing homogenous eosinophilic deposits (individual cells necrosis) with variations of nuclear size and presence of binucleated cells (Stain H&E, X 400).

Figure (4) Suppurative hepatitis: Area of central necrosis enclosed by fibrous capsule, polymorphnuclear and mononuclear inflammatory cells (Stain H&E, X 200).

Figure (5) Parasitic hepatitis: Migrating tracks containing sections of parasites surrounded by eosinophils and mononuclear cells (Stain H&E, X 200).

Figure (6) Parasitic hepatitis: Bile duct hyperplasia with periductal inflammatory cells aggregation mostly mononuclear cells and fibrous connective tissue proliferation with newly formed bile ducts (Stain H&E, X 200).
Figure (7) Parasitic hepatitis: Cystic adenomatous appearance of the bile ducts with the presence of brownish masses inside the lumens of some. (Stain H&E, X 200)

Figure (8) Granulomatous hepatitis: Granuloma consisted of inflammatory cells aggregation mainly mononuclear cells, multinucleated giant cells and fibroblast cells (Stain H&E, X 400)

Figure (9) Granulomatous hepatitis: Granuloma consisted of severe destruction of the bile duct wall surrounded by large number of mononuclear cells and fibrous tissue (Stain H&E, X 400)

Figure (10) Portal cirrhosis: Nodular formation of the hepatic lobules. Note: fibrous connective tissue proliferation enclosed regenerated hepatic cells with absence of central veins (Stain: H&E, X 200)

Figure (11) Portal cirrhosis: Bands of fibrous connective tissue containing numerous blood capillaries and nonfunctional bile ducts and ductules extending between portal regions (Stain Crosman, X 200)

Figure (12) Portal cirrhosis: Porto-portal bridging fibrosis extending between the portal areas (Stain Crosman, X 200)
Figure (13) Portal cirrhosis: Portal tract infiltrated by chronic inflammatory cells with hyperplastic bile ducts and periductal mononuclear cells infiltration and fibrosis (Stain H&E, X 400)

Figure (14) Portal cirrhosis: Thrombus was observed in the portal vein (Stain H&E, X 400)

Figure (15) Biliary cirrhosis: Severely impacted bile ducts by cell debris and inflammatory cells with biliary cells hyperplasia associated with peribiliary mononuclear cellular aggregation (Stain H&E, X 4200)

Figure (16) Biliary cirrhosis: Severe fatty change of most hepatic cells (Stain H&E, X 400)

Figure (17) Biliary cirrhosis: Accumulation of bile pigments in the bile canaliculi forming bile thrombi with atrophy of the hepatic cells (Stain H&E, X 400)

Figure (18) Biliary cirrhosis: Severe accumulation of bile in the hepatic lobules (cholestasis) due to loss of hepatocytes to form lakes of bile (Stain H&E, X 200)
Figure (19) Biliary cirrhosis: Micronodular cirrhosis with replacement fibrosis that enclosing the regenerated nodules (Stain Crossman, X 200)

Figure (20) Central cirrhosis: Marked dilatation and fibrosis of the central lobular vein, with increase in fibrous connective tissue proliferation replacing the hepatic cells around the central vein (Stain H&E, X 400)

Figure (21) Central cirrhosis: extension of fibrous bridges from the pericentral fibrous tissue toward the hepatic lobules (Stain Crossman, X 400)

Figure (22) Central cirrhosis: extension of fibrous bridges from the pericentral fibrous tissue toward the hepatic lobules (Stain H&E, X 400).

Figure (23) Glissonian cirrhosis: Marked thickening of the hepatic capsule due to fibrous connective tissue proliferation (Stain Crossman, X 200)

Figure (24) Glissonian cirrhosis: showing positive PAS glycogen infiltrating granules (Stain PAS, X 400)
Figure (25) Cavernous hemangioma: irregular thin walled channels filled with blood and lined with single layer of endothelial cells (Stain H&E, X 200)

Figure (26) Metastatic lymphosarcoma: Discrete nodules scattered within a portal area showing pleomorphic cells containing pleomorphic vesicular hyperchromatic nuclei with prominent nucleoli and mitotic activities. (Stain H&E, X 200)

Figure (27) Cholangiocarcinoma: Well differentiated glandular and tubular structure lined by anaplastic low columnar epithelial cells, forming more than one cell row with vesicular round to oval, and hyperchromatic nuclei. Variable degrees of mitotic activities. (Stain H&E, X 400)

Figure (28) Cholangiocarcinoma: Glandular structures of varying size and shapes, with hyperchromatic round or oval nuclei and surrounded by dense fibrous stroma (Stain H&E, X400).
Figure (29): Cholangiocarcinoma: the glandular structures surrounded by macrophages, lymphocytes, oval cells and an abundant fibrous stroma. (Stain H&E, X400).

Figure (30): Osteolipomatous metaplasia: Osseous plates formed from osteoblasts and ostoid tissue embedded within the hepatic tissue. (Stain H&E, X 400)

Figure (31): Peliosis hepatis: Dilated blood filled cysts without distinct wall merged with the surrounding liver tissue. (Stain H&E, X 400)

Figure (32): Hepatic telangectasis: Marked dilatation of thin walled blood filled sinusoids. (Stain H&E, X 400)
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