The Camel is generally considered a resistant animal and suffers only a few diseases compared to other livestock species (Narnaware et al., 2021). Hepatocellular injury is one of the pathologic condition affecting domestic animals including camels (Belina et al., 2015). The causes of liver lesions in camel are numerous but primarily liver is affected by toxic substances, infectious diseases, parasitic hepatitis, tumors etc. which contribute to hepatobiliary disease, such as hepatic insufficiency (Kitila, 2016). Mehta et al. (2012) reported maximum mortality (41.39%) in camel due to involvement of digestive system. Björklund (2014) and Tornquist et al. (1991) reported hepatitis and hepatic lipidosis is common pathological findings in camelids. Tharwat et al. (2012) performed ultrasound guided hepatic biopsy to diagnose liver pathology in camels.

Various types of pathological lesions found in liver of camel included hepatitis, cirrhosis (El-Mahdy et al., 2013; Thratw, 2020), fibrosis (Hegazy et al., 2010) necrosis (Abu Damir et al., 1993), non specific hepatic degeneration and fibrosis (Hegazy et al., 2010), liver abscess (Aljameel et al., 2014) and haemosiderosis. Miscellaneous conditions like degenerative changes as fatty changes, hydropic degeneration and various circulatory disturbances as hyperaemia, oedema and haemorrhages are also common. The frequent pathological findings in various diseases of camel comprise of the involvement of liver. In many instances, such lesions in liver are of great value in diagnosis of camel diseases. The pathological conditions of liver in camel have not so far been studied extensively. Thus study was undertaken to find out gross and histopathology of various liver lesions of camels prevalent in Rajasthan.

**Materials and Methods**

For the present study, 80 tissue samples of liver were examined from carcasses of camels of either sex, irrespective of age groups and breeds during post-mortem examination from July 2017 to June 2018. Out of these, 36 samples showed gross lesions were processed for subsequent histopathological examination. An overall incidence of liver lesions in camel recorded was and these 45 per cent (36 out of 80). Diverse pathological lesions in liver were abscess, cirrhosis, fatty changes, haemorrhages, haemosiderosis, hepatitis, hydatidosis, hydropic degeneration and necrosis showing occurrence as 2.50, 8.75, 5.00, 6.25, 1.25, 5.00, 5.00, 3.75 and 7.50 per cent, respectively. Cirrhosis (8.75 per cent) and haemosiderosis (1.25 per cent) were reported as the most prevalent and least prevalent pathological conditions, respectively, affecting liver of camels during the study period.

**Key words:** Camel, liver, lesions, pathology
Results and Discussion

Various liver lesions recorded in present study are placed in Table 1. An overall incidence of various pathological conditions affecting liver was recorded as 45 per cent (36 out of 80) which corresponded well with the findings of Hamza et al (2017) which was 45.7 per cent. Liver showed higher incidence of inflammatory conditions, degenerative changes, circulatory disturbances and parasitic infestation.

Table 1. Incidence of various liver lesions recorded in present study.

<table>
<thead>
<tr>
<th>S. No.</th>
<th>Type of condition</th>
<th>No. of samples</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Abscess</td>
<td>2</td>
<td>2.50</td>
</tr>
<tr>
<td>2</td>
<td>Cirrhosis</td>
<td>7</td>
<td>8.75</td>
</tr>
<tr>
<td>3</td>
<td>Fatty changes</td>
<td>4</td>
<td>5.00</td>
</tr>
<tr>
<td>4</td>
<td>Haemorrhages</td>
<td>5</td>
<td>6.25</td>
</tr>
<tr>
<td>5</td>
<td>Haemosiderosis</td>
<td>1</td>
<td>1.25</td>
</tr>
<tr>
<td>6</td>
<td>Hepatitis</td>
<td>4</td>
<td>5.00</td>
</tr>
<tr>
<td>7</td>
<td>Hydatidosis</td>
<td>4</td>
<td>5.00</td>
</tr>
<tr>
<td>8</td>
<td>Hydropic degeneration</td>
<td>3</td>
<td>3.75</td>
</tr>
<tr>
<td>9</td>
<td>Necrosis</td>
<td>6</td>
<td>7.50</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>36</td>
<td>45.00</td>
</tr>
</tbody>
</table>

**Abscess**

This condition was recorded in 2.50 per cent cases in present study. A higher incidence was reported by Aljameel et al (2014) as 13.5 per cent. Grossly, multiple small white foci containing whitish caseous pus along with congestion and haemorrhages were observed. Microscopically, focal area of necrosis enclosed by thick capsule along with polymorphonuclear leucocytic infiltration (Fig 1) was seen. These changes were in agreement with earlier findings of El-Mahdy et al (2013), Belina et al (2015) and Terab et al (2021).

This condition might be due to pyogenic bacterial infections or fascioliasis and secondary bacterial complication (Belina et al, 2015). Liver abscesses are found in CLA or pseudotuberculosis infection (Terab et al, 2021).

**Cirrhosis**

The occurrence of cirrhosis was 8.75 per cent in present study whereas Jamshidi and Zahedi (2014) found it as 10 per cent.

Grossly, affected liver was reduced in size and was firm and difficult to cut. Microscopically, presence of chronic fibrosed inflammation confined to the areas extending only a short distance beneath the capsule (Fig 2), presence of fibrous bands running in between parenchyma along with little lymphocytic infiltration and perivascular fibrosis mainly in portal area was observed. These findings were in close approximation to the observations reported by Singh (1998) and Ibrahim et al (2021).

Cirrhosis was frequently noticed concomitant with parasitic condition, however, in many cases contributory factors could not be incriminated. Ibrahim et al (2021) reported severe fibrosis in hepatic torsion in camels.

**Fatty Changes**

This condition was recorded in 5.00 per cent cases. A higher incidence was (20%) observed by El-Mahdy et al (2013). Grossly, liver appeared pale, soft and greasy with rounded edges. Histopathologically, the hepatic cells showed fatty changes with presence of fat droplets as clear round spaces and granular cytoplasm (Fig 3) were in agreement with earlier findings of Singh et al (2006) and Zakian et al (2016).

Ketosis may be one of the possible causes of fatty changes. Other factors involved copper sulphate poisoning (Damir et al, 1993) or PPR infection (Zakian et al, 2016) or Brucellosis (Abdirahman, 2020).

**Haemorrhages**

This condition was recorded in 6.25 per cent cases. A lower incidence (2.91%) was reported by Singh (1998). Grossly, large and irregular superficial areas and pin-point haemorrhagic areas of variable sizes were seen on liver. Microscopically, severe haemorrhages replaced the hepatic parenchyma along with congestion, necrosis and presence of inflammatory infiltration in adjacent parenchyma were in agreement with observations of Singh (1998) and Ibrahim et al (2021).

This condition might be due to trauma or Aflatoxin B1 residues (Al-Gabri, 2013) or hepatic torsion (Ibrahim et al, 2021).

**Haemosiderosis**

This condition was recorded in 1.25 per cent cases which corresponded well with the findings of Singh (1998) who found it 1.45 per cent. Grossly, the liver showed brownish tinge due to the presence of haemosiderin in hepatic cells. Microscopically, haemosiderin as golden coloured pigment filled in phagocytes (Fig 4) was in agreement with Singh (1998) and Ibrahim et al (2021).

This condition might be observed in scars, in areas where haemorrhages had occurred or in chronic
Fig 1. Microphotograph of liver abscess showing focal area of necrosis enclosed by thick capsule along with polymorphonuclear leucocytic infiltration. H&E 100X.

Fig 2. Microphotograph of liver showing red coloured fibrous tissue along with thickened glisson’s capsule. Van Gieson’s 100X.

Fig 3. Microphotograph of liver showing fatty changes indicating fat droplets as clear round spaces. H&E 200X.

Fig 4. Microphotograph of liver showing haemosiderin as golden coloured pigment filled in phagocytes. H&E 400X.

Fig 5. Microphotograph of liver showing multiple cysts with eosinophilic lamellated wall and presence of faint pink homogenous substance along with hepatocellular degeneration. H&E 100X.

Fig 6. Microphotograph of liver showing hydropic degeneration as small clear vacuoles along with cellular infiltration. H&E 200X.

**Hepatitis**

This condition was recorded in 5.00 per cent cases. A higher incidence (20%) was reported by El-Mahdy et al (2013). Grossly, the liver was markedly enlarged and centres of lobules were opaque, grayish or yellowish. Microscopically, infiltration of lymphocytes and few neutrophils along with areas of hepatocellular degeneration was seen. These changes were in agreement with earlier findings of Seboussi et al (2009), El-Mahdy et al (2013) and Tavella et al (2018).

The inflammation of liver might be caused by various conditions including toxicity of diminazene aceturate (Homeida et al, 1981) or selenium toxicity (Seboussi et al, 2009).

**Hydatidosis**

This condition was recorded in 5.00 per cent cases. Almost similar findings were reported by Mirzaei et al (2016) as 4.54 per cent. Grossly, grayish white detectable hydatid cysts of variable sizes were located either single or multiple on the surface and embedded in the hepatic parenchyma which was in agreement with earlier report of Osman (2008). Microscopically, cyst wall appeared as outer eosinophilic lamellated layer with presence of faint pink homogenous substance within the lumen (Fig 5) was in harmony with those mentioned by Tantawy (1992) and Borai et al (2013).

Hydatidosis in liver might be due to the fact that liver possesses great capillaries site encountered by the parasite, which adopt the portal vein route and primarily hepatic filtering system sequentially (Kebede et al, 2009). Nourani and Salimi (2013) reported hydatid cyst as the most common hepatic lesion in liver of camels.

**Hydropic degeneration**

This condition was recorded in 3.75 per cent cases. A higher incidence (18.67%) was recorded by El-Mahdy et al (2013). Microscopically, the liver cells increased in size due to accumulation of fluid. Small clear vacuoles seen within the cytoplasm along with hepatocellular degeneration and cellular infiltration (Fig 6) which were in agreement with Singh et al (2006), El-Mahdy et al (2013) and Tavella et al (2018).

This condition might occur due to pathogenic infections such as Hepatitis E virus 8 infection (Wang et al, 2018) or toxic conditions such as closantel toxicity (Raval et al, 2018).

**Necrosis**

This condition was recorded in 7.50 per cent cases and a higher incidence (18.1%) was reported by Salem and Azza (2011). The gross findings were presence of grayish necrotic foci on surface. The involved hepatocytes showed dense, granular and opaque cytoplasm with coagulative necrosis. Similar findings were reported by Borai et al (2013) and Zakian et al (2016).

This presumably may be due to involvement of some toxins in feed possibility fungal, bacterial or plant toxins or chemicals playing some role in causing the condition.

Groom et al (1995) reported hepatic-necrosis in an alpaca which was associated with halothane anaesthesia possibly due to hepatotoxic effect of halothane on liver.

In present study the tumours of liver were not seen. However, intrahepatic cholangiocarcinoma in an 18-year-old male camel has been reported and it was composed of gland-like structures and/or solitary islands of neoplastic cells in the tumoral stroma (Birincioglu et al, 2008).

Most of the pathological conditions of liver in camels are diagnosed during postmortem examination. However, a more detailed study of liver lesions is required to identify these pathological conditions during clinical examination through modern diagnostic tools.

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