Vol 20 No 1, p 97-100

PATHOLOGICAL STUDY ON LIVER OF DROMEDARY CAMELS

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ABSTRACT

This study was undertaken to find out gross and microscopic hepatic lesions of dromedary camels slaughtered in the central part of Iran. The grossly affected livers from 156 camels with different ages and unknown history were collected from freshly slaughtered animals at Najaf-Abad abattoir, Iran and examined grossly and histopathologically. The pathological changes observed were caseous necrosis and calcification (18.58%), hydatid cysts (12.17%), lipomatous lesion (3.2%), hepatocellular degeneration and pigmentation (5.1%), toxic hepatic lesions (1.92%), liver atrophy and fibrosis (1.28%), fascioliasis (1.28%), focal hepatic lipidosis, *Corynebacterium pseudotuberculosis* lesions, and haematoma, each one (0.64%). In the present study, focal caseous necrosis and hydatid cysts were the most common hepatic lesions in the examined camels. In some liver sections, partially degenerated parasitic ova and hydatid cysts were identified as cause of focal caseous necrosis and calcification.

Key words: Dromedary camel, Iran, liver pathology

There are several methods that have been used to diagnose hepatic disorders. In general, these methods involve the evaluation of clinical history, physical examination, biochemical tests, hepatic imaging, gross and histopathological examinations (Al-Sobayil, 2008).

In previous studies, various pathological lesions including hydatid Cysts (Ahmadi, 2005), *Fasciola hepatica* associated lesions (Eslami *et al,* 2003), linguatulosis (Haddadzadeh *et al,* 2009; Oryan *et al,* 2011; Shakerian *et al,* 2008), abscesses of *Corynebacterium pseudotuberculosis* (Hawari, 2008), multicentric T-cell lymphoma (Simmons and Fitzgerald, 2005), Peripheral primitive neuroectodermal tumour (Weiss and Walz, 2009) and Multicentric fibromyxoid peripheral nerve sheath tumor or multicentric schwannoma (Khodakaram-Tafti and Khordadmehr, 2011) have been described in the liver of camels. This study was undertaken to find out gross and microscopic hepatic lesions of dromedary camels slaughtered in the central part of Iran.

Materials and Methods

The grossly affected livers from 156 dromedary camels with different ages and unknown history were collected from freshly slaughtered animals at Najaf-Abad abattoir in the central part of Iran and examined carefully for macroscopic lesions. For histopathological study, tissue samples were taken from the hepatic lesions and fixed in 10% neutral buffered formalin. These were processed and embedded in paraffin. Sections of 5 µm thickness were cut and stained with haematoxylin and eosin.

Results

In this study, various gross and microscopic lesions were observed in the liver of 156 examined camels that are summarised in Table 1.

Table 1. Pathological changes observed in the liver of 156 examined camels.

Pathological changes	Number	Per cent
Caseous necrosis and calcification	29	18.58
Hydatid cysts	19	12.17
Lipomatous lesion	5	3.2
Hepatocellular degeneration and pigmentation	8	5.1
Toxic hepatic lesions	3	1.92
Liver atrophy and fibrosis	2	1.28
Fascioliasis	2	1.28
Focal hepatic lipidosis	1	0.64
Corynebacterium pseudotuberculosis lesions	1	0.64
Haematoma	1	0.64
Total	71	45.51

Caseous necrosis and calcification was the most common hepatic lesion seen in 29 camels (18.58%). They had various size and degree of calcification.

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Hydatid cysts were observed in 19 camels (12.17%). The number of cysts varied from one to fifteen and their size ranged from smaller than one to nine centimetres. Grossly and microscopically, some of the cysts showed evidence of degeneration and had thicker wall, caseous necrosis and calcification.

Lipomatous lesion was identified in 5 cases (3.2%). Grossly, these lesions comprised of one or two white coloured nodules, up to 1 cm in diameter, circumscribed but not encapsulated and had irregular borders. Microscopically, the nodules consisted of aggregations of adipocytes resembling normal fat cells (Fig 1).

Hepatocellular degeneration and pigmentation were detected in 8 camels (5.1%). Vacuolated hepatocytes had yellow to brown and granular pigments (Fig 2).

Toxic hepatic lesions were observed in 3 camels (1.92%). Histopathological examination of the affected livers revealed fatty change, vacuolated giant hepatocytes (megalocytosis, Fig 3), hepatocellular pigmentations, hyperemia and haemorrhages.

Liver lobe atrophy was seen in 2 camels (1.28%). Microscopically, the atrophic lobe consisted of diffuse fibrosis, hyperplastic bile ducts, and some foci of inflammatory cells.

Fasciola spp. associated lesions were found in 2 camels (1.28%). The lesions were due to adult flukes and grossly consisted of bile ducts ectasia (Fig 4) with numerous mature *Fasciola* spp. within them, discolouration of liver lobes, focal accumulations of brown to black pigments, severe thickened bile ducts and foci of caseous necrosis. Histopathological examinations of the parasitic lesions revealed adult flukes within bile ducts, biliary epithelial cell necrosis and desquamation, infiltration of inflammatory cells or cholangitis, glandular hyperplasia of dilated bile ducts, severe periductular fibrosis, bile duct hyperplasia and diffuse biliary epithelial proliferation that has obliterated liver parenchyma in many foci.

Variable numbers of viable or calcified parasitic ova were present in the lumens of affected bile ducts and in the connective tissue adjacent to the ducts (Fig 5). Partially degenerated parasitic ova were observed in the centre of some foci with caseous necrosis and calcification too.

On microscopic examination, focal lipidosis was observed in liver of one camel (0.64%) adjacent to attached pancreas.

The various size abscesses of *Corynebacterium pseudotuberculosis* were found in one camel (0.64%) that randomly scattered in liver lobes. Incision of these abscesses revealed thick, whitish, caseated, and lamellar material (Fig 6). Microscopically, consecutive layers of caseous necrosis and calcification were surrounded by fibrous wall.

There was a haematoma, 1 cm in diameter, on hepatic surface of a camel. Microscopically, the hematoma consisted of red blood cells and fibrin strands in centre that were surrounded by inflammatory cells and connective tissue.

Discussion

The diagnosis of hepatic lesions through the histopathological evaluation is a very useful method. It can be performed after taking a liver biopsy or post mortem sampling. Liver histopathology can often define the liver diseases as infectious, toxic, or obstructive and congestive. It is usually performed to diagnose hepatic tumours or to evaluate the extent of damage that has occurred to the liver because of chronic diseases (Al-Sobayil, 2008).

In the present study, the infection rate of hydatid cyst was 12.17% in liver. In our previous study about pathological changes of lungs, a higher infection rate of hydatid cyst (51%) was found in lungs of examined dromedary camels in this region (Nourani and Rohani, 2009). Various infection rates of hydatidosis have been reported from other countries including Pakistan (40%, Zubair *et al,* 2004), Kuwait (39.6%, Abdul-Salam and Farah, 1988), Libya (35.9%, Gusbi *et al,* 1990), Morocco (14.3%, Tligui *et al,* 2006) and Egypt (7.67%, Dyab *et al,* 2005).

In the present investigation, lipomatous lesion was identified in 5 cases (3.2%). Similar lipomatous lesions have been reported in the livers of dromedary camels, a bactrian camel and some new world camels that spicules of mature bone were scattered throughout the nodules of a few animals (Stroud *et al,* 1982) but in our study, there is no bone formation. Metaplastic changes of pluripotent connective tissue cells are proposed as cause of adipose tissue and bone formations in parenchyma of the camel liver (Stroud *et al,* 1982).

Our findings revealed that 1.28% of the examined livers had *Fasciola* spp. lesions. Eslami *et al* (2003) reported a prevalence of 5.3% for *Fasciola hepatica* in dromedary camels slaughtered at industrial abattoir of Mashhad, north west of Iran. In this study, partially degenerated parasitic ova were observed in the centre of some foci with caseous necrosis and calcification. Similar finding has been reported

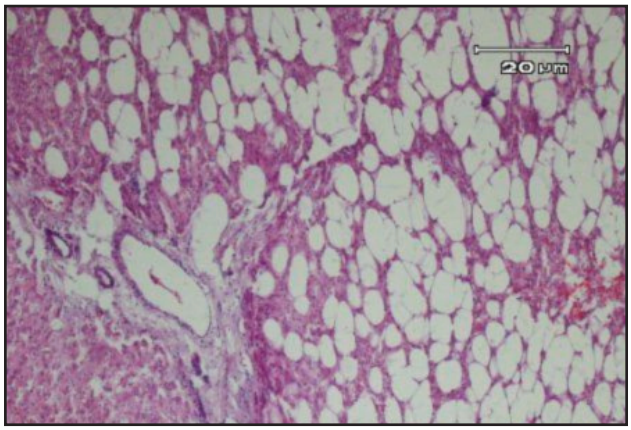

Fig 1. Lipomatous lesion. Note aggregation of adipocytes adjacent to a portal area (H &E, X 100).

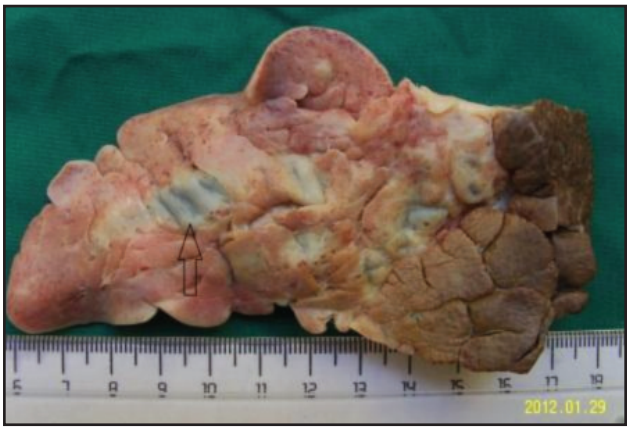

Fig 4. Bile ducts ectasia (arrow) due to adult flukes.

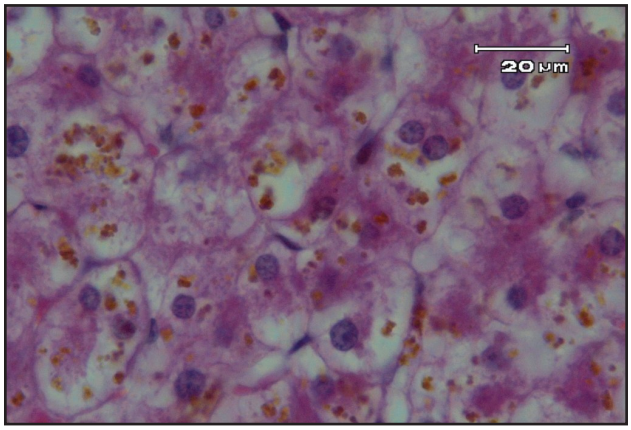

Fig 2. Hepatocellular degeneration and pigmentation (H &E, X 1000).

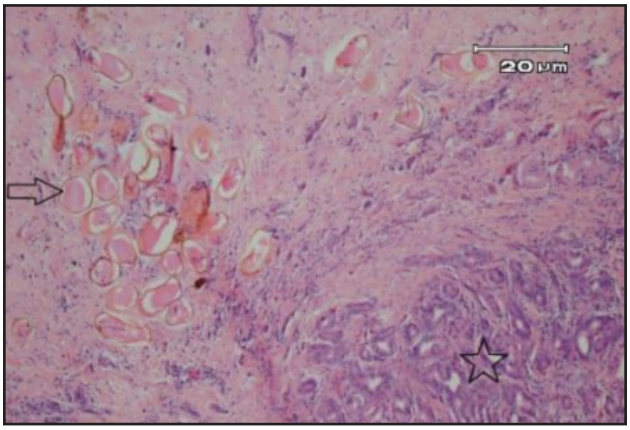

Fig 5. Liver fluke ova (arrow) are embedded in periductular fibrosis; and glandular hyperplasia (star) of bile duct. (H &E, X 100).

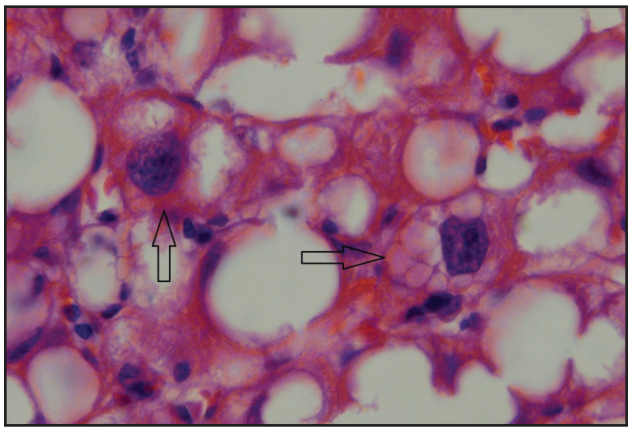

Fig 3. Toxic hepatic lesion, megalocytosis. Two vacuolated giant hepatocytes (arrows) are seen (H &E, X 1000).

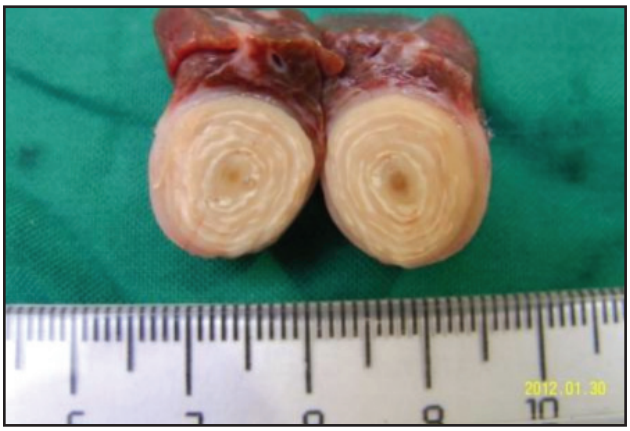

Fig 6. A dried abscess of *Corynebacterium pseudotuberculosis*, cut section. Consecutive layers forming the typical pattern of what is called onion-like appearance.

by Hamir and Smith (2002) in a case of liver fluke infection in an adult alpaca.

In our study, hepatic lipidosis or fatty change was observed adjacent to attached pancreas and together with toxic hepatic lesions. Fatty change and necrosis of the liver cells have been observed in experimental copper poisoning of dromedary camels (Abu Damir *et al,* 1993).

In this study, abscesses of *Corynebacterium pseudotuberculosis* were found only in the liver of

one camel. Hawari (2008) has reported multiple muscles, subcutaneous, and internal organs abscesses of *Corynebacterium pseudotuberculosis* in dromedary camels of Jordan.

In the present study, focal caseous necrosis and hydatid cysts were the most common hepatic lesions in the examined camels. In some liver sections, partially degenerated parasitic ova and hydatid cysts were identified as cause of focal caseous necrosis and calcification.

Acknowledgement

This study was supported by Research Council of the Shahrekord University, Shahrekord, Iran. The authors are grateful to Mr. Ahmadi for slides preparations.

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