NON SPECIFIC HEPATIC DEGENERATION AND FIBROSIS IN CAMELS (CAMELUS DROMEDARIUS)

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ABSTRACT

888 slaughtered camel of both sex were investigated for the presence of hepatic affections. The results showed that about 15% of the livers were partially or totally condemned for various reasons including: echinococcosis, abscesses, multifocal necrosis, hepatic congestion and /or fibrosis. The latter constituted 70% of the affections and some of these cases were associated with ascitis and subcutaneous edema with no detectable gross abnormalities in other visceral organs. Microscopical examination showed three types of lestons, a) congestion and early degeneration (70%), b) advanced vacuolar degeneration, early necrosis and sinusoidal congestion (20%), c) necrosis and advanced fibrosis accompanied by billary epithelium hyperplasia (10%). No specific pathogen, parasite or heavy metals have been yet identified. However, the role of environmental toxins could not be rule out.

Key Words: *Liver, camel, hepatic, degeneration, fibrosis, bacteria, parasites, toxins.*

INTRODUCTION

Liver plays an important role in metabolism. It is exposed to various toxic metabolic as well as pathogens and parasites **(Afzal 1995)**. Limited aspects of camel liver pathology have been studied. Liver fasciolliasis **(Eisen 2000; Haridy 2000; Mahmoud et al. 1989; Fahmy and El-Ataar 1990 and Farah 1993)**. cysticercosis **(Al-tabari 2009; Eisen 2000)** and hydatidosis **(Ahmadi, 2004; Altabari 2009; Tashani et al 2002; Zarffard 2000; Tantway 1992 and Al-Rashed et al. 1994)** were reported.

Liver abscess have been reported by **Altabari (2009)**, who isolated Corynebacterium pyogencs, *E. coli* and *Pseudomaius aeruginosa* as the most common incriminated micro-organism.

Antimicrobial resistance pattern of Salmonella serotypes have been reported **(Bayleyegn et al 2004; Wernery 1992)**.

El Naenaeey (2000); Makhareta (1988) and Ithman et al. (1989) found that *Clostridium novyi, Clostridium perfringens* and *Fusibacterium necrophorum* were the most important anerobic micro-organism isolated from camel liver abscesses or necrosis.

The importance of plant secondary metabolites in ecology, human foods and animal feeds, and as pharmaceutcals with chemical and biochemical attributes has already been described in some detail **(D'Mello 1997; Brooker 2000; Harborne 2001; Pfannhauser et al 2001; Acamovic et al 2004; Nash 2004).**

The aim of this paper is to report the hepatic lesions in camel in the Eastern Region of Saudi Arabia and to throw light on its incidence, pathological profile, and possible causes with special reference to liver degeneration and fibrosis.

MATERIALS AND METHODS

688 local camels of both sexes and different ages were examined for detection of hepatic diseases. The liver as well as the carcasses and visceral organ were examined grossly for presence of any abnormalities. Liver samples from different lesions were collected for bacteriological, toxicological and pathological examinations.

Bacteriological Investigation :

Samples were aseptically taken from the freshly slaughtered camels: each sample was placed in a sterile plastic bag, kept in an ice bag and sent to the laboratory within few hours after sampling. The surface of the liver tissue was sterilized using hot spatula and a bacteriological swab taken from the periphery of lestons, if confined, or from the cirrchotic hepatic tissue in case of diffuse hepatic cirrhosis. Each sample was streaked onto: Hektoen enteric (H.E) Agar, Blood Agar (B.A) Reinforced Clostridial Medium (R.C.M.) and Sabourauds's Dextrose Agar (S.D.A.). The inoculated H.E. and B.A. plates were incubated aerobieally at 37%°C, while R.C.M was incubated anaerobically at the same temperature. S.D.A. inoculated plates were incubated at room temperature (25°C ± 2°C). Plates were then examined for visible colonies 24 hours after incubation and then daily for 5 consecutive days. Plates that did not reveal any visible colonies were then discarded as negative. The growing colonies were picked up, cultured on slope agar tubes incubated again at 37°C for 24 hours and were subjected to biochemical identification using the API 20E system (API Analytical Profile Index, BioMerieu, France). The bacteriological examination and identification of the recovered organisms were done according to balows et al. **(Downes 2004, Dwightc 2004 ,Jay 2006).**

Toxicological Investigation :

Parts from cirrhotic and congested livers (50 g each) were collected and stored at -20°C till analysis.

Histopathological Investigation :

Liver samples were fixed in 10% neutral formalin. 3-5μ paraffin sections were prepared and stained by haematoxylin and eosin (H&E).

Detection of aflatoxins in the samples was performed according to **AOAC (1995).** Copper and selenium detection was conducted according to **Timberll (1992); Wernery (2002)** using dithizone method and Reinsch test respectively.

RESULTS

The macroscopical examination of 688 liver revealed that 1103 livers had variable lesions.

The type and incidence of the lesions are shown in Table 1.

Bacterial Findings :

E. coli was isolated from almost all samples of congestion and for cirrhosis. No strict anaerobic bacteria or fungi could be isolated. Streptococcus faecalis, Pseudomonas aeruginosa and *E. coli* were isolated from abscesses.

Toxicological Findings :

All the investigated samples were free from aflatoxins and no toxic levels of copper and selenium could be detected.

Pathological Findings :

1. Echinococcosis : 29 lives showed variable numbers of hydatid cysts embedded in the parenchyma or protruded on the surface. The cyst consisted of 2 layers, an outer thick layer creamy in colour and inner germinal layer to which scolices and broad capsule were attached. Some cysts were caseated and calcified (Fig. 1). Microscopically: The thick wall appeared lamellated and surrounded by lymphocytes, eosinophils and round cell infiltrations. The adjacent hepatic cells were atrophied.

2. Abscesses : This formed about 0.2%. Only two livers had abscesses. One showed a single large abscess 5 cm in diameter surrounded by thick connective tissues capsule and contained creamy caseated material and other had multiple abscesses associated with extensive easeous necrosis and fibrosis (Fig. 2). Microscopically, the abscesses showed central liquifactive necrosis surrounded by pus cells and fibrous capsule. The surrounded liver cells showed degenerative changes with portal aggregations of round cells and few neutrophils.

3. Focal Hepatic Necrosis : Grossly the lesions were multiple, in the form of grayish foci or irregular patches. They were sub capsular or embedded in the parenchyma. The livers were slightly enlarged. Microscopically the lesions appeared as areas of coagulative necrosis surrounded by and infiltrated with variable numbers of round cells (Fig. 3), and few neutrophils. E coli type 1, Klebsiella, was isolated from these cases. No strict anaerobes could be isolated.

4. Hepatic Congestion : 48 livers showed marked congestion. It was observed more frequently during the hot summer season (75%). The livers appeared enlarged, about two-fold its normal size. It was purplish in colour with marked accentuated hepatic lobules (Fig. 4). On incision the lesions exhibited a reticular like structure filled with blood. Microscopically marked centre lobular congestion associated with cellular atrophy of the central zone and vacuolar degeneration of the peripheral zone were the predominant pathological features. In some cases the congestion was so marked as to give the appearance of telangiectasis. In advanced cases extensive parenchymal haemorrhages was seen associated with marked cellular necrosis (piliosis).

5. Advanced Parenchymal Degeneration: The liver appeared enlarged mottled brownish in colour. On cut section dark red to reddish purple areas surrounded by paler parenchyma were the pronounced pictures (Fig. 5). Microscopically, diffuse areas of hepatic degeneration were persistently observed. Small

areas of congestion or even haemorrhages were seen. Multiple focal hepatic necrosis associated with variable degrees of portal and pericellular fibrosis and inflammatory cell infiltration were observed

6. Hepatic Fibrosis: Grossly the lesions were focal involving several areas except in one case where the fibrosis was diffuse. The affected areas appear firm, with irregular nodular surface occasionally associated with marked thickening of hepatic bile ducts. The microscopic picture of the fibrosed livers varied from chronic persistent hepatitis was characterized by portal fibrosis, with excessive rounds cells infiltration and fibroplasias with preservation of hepatic cell plate. The chronic aggressive hepatitis was characterized by hepatic cell necrosis especially those of the hepatic cell plate associated with extensive fibrosis replacing the necrotic cells and invading the hepatic lobules (Fig. 6).

In diffuse hepatic cirrhosis the hepatic tissue was replaced by massive fibrous tissue encircling few islands of atrophied or even necrotic hepatic tissue. Adenomatous changes of bile ducts were clearly detectable. The ducts were cystically dilated, lined with tall columnar or low cuboidal epithelial cells, with faint bluish cytoplasm and basely located hyperchromatic nuclei. Mild dysplasia was noticed.

DISCUSSION

The present investigation shows that about 15% of camel livers may have various pathological affections of parasitic, bacterial or undefined etiology. Considering the large numbers of camels slaughtered annually for human consumption (500 animals in Al-Ahsa abattoir alone) the losses are economically sizable. In addition, some liver affection are associated with generalized edema and poor carcass condition which occasionally lead to total carcass condemnation, thus adding to economical losses.

The only parasitic condition observed in this study was due to echinococcosis which constituted 4.2% of the total liver affections. Echinococcosis appears to be common in local camels and other domestic ruminants **(Dinkel; 2004; Eisen 2000; Farah, 1993; McManus 2003)**.

Only two cases of liver abscesses were recorded in this study suggesting that hepatic pyogenic infections are rather uncommon in camels. However, the recovery of Aeromonas hydrophila from a case of hepatic abscess in a camel could be of significance because this organism has been isolated from diseased domestic and zoo animals **(Carter and Cole, 1990)** who reported that the isolation of this organism from animals is scant, but on occasions it can account for infections in animals. Similar findings have been reported by **Tejedor (2004); Makhareta1 (1988) and Ithman et al. (1989)**. Hepatic abscesses may occasionally and fatally in camels.

The hepatic congestions, degenerative and necrotic changes and cirrhosis currently observed seen to constitute successive stages of progressive disease condition which starts with congestion and ends with extensive fibrosis. These changes are difficult to explain in

the absence of a definite etiology **(Gameel et al. 2003)**. Hepatic necrosis and fibrosis have been reported in camels slaughtered in Bureida (Saudi Arabia); some were related to parasitic and bacterial infectious and others were attributed to unknown faetors However, hepatic congestion in animals is an expected sequel of right-heart failure. No signs of congestive heart diseases or hepatic vein occlusion could be detected. Hence, the few cases of anasarca noticed could be related to decreased concentrations of plasma proteins resulting from hepatic cirrhosis.

Congestion with venous occlusion, necrosis and fibrosis have been reported in livers of bovines and equines and were related to toxicity with pyrolizidine alkaloids which are found in many plants belonging to the genera Senicio, Cortaloria, Triholuma and others **(Acamovic 2005; Copper and Johnson 1984; Mohneux et al. 1991; pearson 1991)**. However, non of these plants are known to prevail in natural camel habitat in Saudi Arabia.

It is known that toxic hepatitis can be induced by aflatoxins **(Peterson 1982)** and liver cirrhosis can be caused by various chemical poisons including copper and selenium **(Wernery 2002)**. Chemical analysis of the affected livers under study, failed to indicate any of these as a cause of necrosis or fibrosis. On the contrary there is a strong evidence that copper and selenium deficiency occurs in this area **(Diab et al. 2003; Ali and Al-Noaim 1991)**.

However, The zinc and eopper content of the plasma of Sudanese camels(Camelus dromadarius), have been reported by **Mohamed (2004)** In conclusion, the etiology of the degenerative and neerotic hepatic lesions in camels needs to be thoroughly investigated. We believe that toxic plants, industrial toxins and various adverse environmental factors may be involved in condition.

Table 1. Types and incidence of hepatic lesions.

Lesion	No	Incidence	Remarks
Echinococcosis	29	4.0%	Cysts seen in lungs
Liver abscess	2	0.3%	Streptococcus Faecalis E. coli, Aeromonas Hydrophila isolated.
Congestion	48	7.0%	Mostly in summer, 16 cases showed Ascitis And anasarca
Focal Necrosis	6	0.9%	E.coli type 1 was isolated
Partial Fibrosis	7	1.0%	3 cases showed ascitis and edema
Diffuse Fibrosis	1	0-15%	Ascitis and Edema
Total	**93**		

Fig. 1 : Liver showing two hydatid cysts.

Fig. 3 : Liver: Showing coagulation necrosis with round cell infiltration and fibroblasts proliferation. H & E X 100.

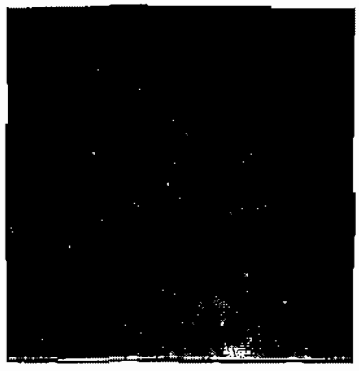

Fig. 2 : Liver: Multiple abscesses.

Fig. 4 : Liver congestion with marked accentuated hepatic lobules.

Fig. 5 : Enlarged mottled brownish liver.

Fig. 6 : Extensive hepatic fibrosis with biliary Adenomatous changes. H & E x 100.

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