

PATHOLOGICAL STUDIES ON HEPATIC LESIONS OF CAMELS (*Camelus dromedarius*) SLAUGHTERED AT TAMBOUL ABATTOIRS, SUDAN

Mohammed H. Al-Hadi¹ & Amir M. Saad²

1. Department of Pathology, Faculty of veterinary sciences Nyala University
2. Department of Pathology, Faculty of Veterinary Medicine University of Khartoum

المستخلص

اجريت هذه الدراسة لمعرفة الاسباب المرضية لاعدام اكباد الجمال التي ذبحت بمسلخ تمبول. فحصت في هذه الدراسة (100) كبد معدمة عيانيا ومجهريا باستعمال صبغة الايوزين والهيماتوكسلين. استعملت اصباغ خاصة للتعرف علي انواع الافات والمسببات المرضية. تم تشخيص الحالات المرضية الاتية : الاكياس العدارية (33%)، التشمع الكبدي (15%)، النخر (13%)، الاحتقان (13%)، الخراجات (9%)، التغيرات الدهنية (7%)، النزف وتوسع الجيبانيات الكبدية (5%)، التكلس (3%)، الالتصاقات (2%)، نم الوصف النسيجي المرضي للحالات المذكورة واوضحت الصبغات الخاصة وجود بعض المستعمرات البكتيرية الموجبة لصبغة الجرام من النوع العصوي والكروي في بعض القطاعات النسيجية التي اخذت من الخراجات وذلك عند صبغها بجرام. اما الألياف الغروية في حالات التشمع الكبدي فقد ظهرت بلون احمر عند استعمال صبغة فان جيسون.

Abstract

This study was carried out to investigate the pathological causes of camel livers condemned at Tamboul slaughterhouse . A total of 100 condemned livers of camels at Tamboul abattoir were grossly and microscopically examined using routine H&E stain. Special stains were furtherly used to verify the lesions and identify organisms. The pathological conditions as judges macroscopically included hydatid cysts (33%), cirrhosis (15%), necrosis (13%), congestion (13%), abscess (9%), fatty changes (7%), telangiectasis&haemorrhages (5%), calcification (3%) and adhesions (2%). Histopathological examination was made to describe the microscopic picture of these conditions. Gram positive colonies of bacteria (cocci and bacilli) were demonstrated using gram's stain in sections taken from abscesses. For

demonstration of collagen fibers particularly in liver cirrhosis conditions, Van Gieson's stain was used and the fibers were stained by deep red color.

Introduction

Camel (*Camelus dromedarius*) is an important multipurpose animal in arid and semi-arid areas of the world. (FAO, 1992). It is kept for a variety of purposes e.g. transportation, racing and as source of human food (Dorman, 1986). Sudan is one of the largest camel populated countries in the world. According to FAO statistics, camel population in Sudan ranks the second in the world after Somalia with 4.5 millions heads. (Faye *et al*, 2011).

Affections of the liver constitute a major factor that reduces our national income, either directly through condemnation of the affected livers, or indirectly by their effect on the animal growth and so its meat production (Pratt, 1992). In camelids, liver disease has not been widely recognized. Although fatty liver disease is often observed during necropsy examination. Primary liver diseases have been infrequently diagnosed (van Saun *et al*, 2000). Livers are usually eaten raw by many communities in Sudan, because if they are cooked, they become elastic and inedible. Therefore, camel liver is potentially hazardous. Yet, in spite of that it is still regarded as a very popular dish. (Abdel-Rahman, 1996). At the slaughterhouses, a number of specific and non specific liver conditions might be diagnosed and some of them are important because of their zoonotic nature and epidemiological importance. In this context this study was conducted at Tamboul slaughterhouse to identify causes of liver condemnations in slaughtered camels at Tamboul area and study and elucidate the gross and histopathological picture in most commonly identified lesions, demonstrate some causative agents using routine and special staining techniques.

Material and Methods

Study area:

This study was conducted at Tamboul locality, Eastern Gazira Region, 150 km South of Khartoum North. It is the capital town of Butana area, and the largest camel market in Sudan.

Animals:

100 livers from slaughtered camels of both sexes and different ages in Tamboul slaughterhouses were used

Tissue specimens:

After slaughtering the animals, careful gross examination of the livers was carried out. Gross lesions were recorded. Then tissue samples were taken, fixed in 10% neutral buffered formalin, dehydrated, cleared and embedded in paraffin. Sections of the embedded tissues in the wax blocks were cut by a microtome, using disposable blades, to get sections of 4-6 μ in thickness.

Staining:

Paraffin sections were routinely stained with Hematoxylin and Eosin, Moreover, the following special stains were carried out in an attempt to demonstrate the causative agents or for verifications of the lesions, according to RAB and Wallington (1980)., Bancroft et al, (1996)

- 1-Gram's technique for bacteria in sections.
- 2- Ziel-Neelsen technique for acid-fast bacilli.
- 3- Periodic Acid-Schiff (PAS) reaction for demonstration of fungi and carbohydrates in sections.
- 4-Van Gieson technique for demonstration of collagenous fibers.

Results

Causes of liver condemnation:

The pathological causes of condemnation of 100 camel livers in Tamboul slaughterhouse as judges by gross lesions are shown in Table 1.

Macroscopic and microscopic appearance:

Hydatid cysts constituted the highest percentage of condemnations as compared with other causes. 33 livers were condemned due to hydatidosis. Macroscopically the sizes of cysts varied from one to fifteen cm in diameter and were found embedded in liver tissue, mostly bulging or protruding from the surface. (Fig 1) Histopathologically, thick cyst wall of fibroelastic connective tissue associated with severe infiltration of lymphocytes and eosinophils were seen. The pericystic hepatic tissue showed dilated sinusoids with atrophied hepatocytes. Violet irregular calcium granules were present in some hydatid cysts. (Fig 2). Cirrhosis was found in 15 livers as the second cause of condemnation. On gross examination, diffuse fibrosis resulting in disorganization of the liver lobules were seen. Histopathological examination revealed massive fibrosis interspersed with regenerated hepatic lobules and necrotic hepatocytes with architectural distortion of the hepatic lobules. Sometimes fibrous tissues extended to make pseudo lobulations and the lobules were atrophied. The collagen fibers stained deep red with Van Gieson's stain. While the parenchyma stained bright yellow.

Thirteen livers (13%) of total condemned livers were due to hepatic necrosis. Macroscopically the lesions varied from focal to multi-focal, caseated and calcified lesions, different in color (dark, pale, whitish ect), size, usually numerous, randomly distributed on sub-capsular region and in the parenchyma. (Fig 3). Histopathologically the lesions appeared as focal parenchymal necrosis around the central vein, portal areas in addition to the mid-zone and in some cases it may involve large areas of liver lobules centre (centrolobularly). Some necrotic lesions were associated with fatty changes (Fig.4).

Nine livers (9%) were condemned due to liver abscesses. Grossly the livers had multiple well-demarcated coalescing yellowish to whitish foci with a caseous consistency in some livers and pasty, either embedded in or bulging from the surface of the liver and were few to several centimeters in diameter, surrounded by capsules that vary in thickness. (Fig 5). Microscopically, abscesses were composed from the periphery to the centre outer zone of

fibrous capsule consisting of fibrocytes, collagen, and elastic fibers, pyogenic membrane formed mainly of neutrophils and the liquefactive necrotic area in the center. In some abscesses heavy infiltration of the neutrophils in focal necrotic areas were seen. In others there was calcification in the center and in one case associated with fatty changes. (Fig6).

Seven livers (7%) were condemned due to fatty changes. Grossly fatty liver was enlarged and showed pale yellowish colour with greasy appearance, rounded edges and the cut surfaces bulged when incised. Microscopically microvesicular and macrovesicular vacuolar change which alters contour of hepatocytes and displaces the nucleus, when stained with (H&E). In case of hydropic degeneration the hepatocytes were swollen and contained small vacuoles without sharp border, when stained with (H&E).

Thirteen livers (13%) were condemned due to hepatic congestion. Grossly congested livers appeared dark red or blackish sometimes with other lesions such as abscesses, haemorrhages and telangiectasis. (Fig 7). Microscopically congested livers revealed blood vessels congested with erythrocytes. The portal areas were necrotic, markedly dilated and filled with erythrocytes. (Fig 8). Five livers (5%) were condemned due to haemorrhages and hepatic telangiectasis. Grossly petichial or echymotic haemorrhages were observed confined to one lobe or distributed in both hepatic lobes. These condemned livers were also associated with hepatic telangiectasis, they were grossly characterized by single or multiple red-brown foci. Microscopically areas of dilated and congested sub-capsular sinusoids with large numbers of erythrocytes were observed in sections, while most of the hepatocytes were atrophied, degenerated or necrotized.

Three livers (3%) were condemned due to calcification. Grossly, calcification appeared as white or yellowish gritty areas varying in size from few millimeters to one or more centimeters in diameter and mostly scattered in the liver parenchyma with gritty feeling when the tissue were sliced with a knife. Microscopically Irregular violet calcium granules were observed in liver tissue when stained with H&E.

Two livers (2%) were condemned due to adhesions. Grossly numerous pale fibrous tags on the diaphragmatic surface of the liver. Histopathological sections revealed thick fibrous layers adherent to liver tissue. Gram's stain revealed presence of gram positive colonies of bacteria (some of them gram+ve cocci and others bacilli) in some sections prepared from abscesses (Fig. 9 &10).Periodic acid Schiff and Zeil Nelsens stains did show any fungi or acid fast organisms

Table: (1): Condemnation percentages of camel's livers due to various pathological conditions.

Causes of Condemnation	Numbers of Condemned Livers	Percentage of condemnation
Hydatid cysts	33	33
Cirrhosis	15	15
Necrosis	13	13
Abscesses	9	9
Fatty changes	7	7
Congestion	13	13
Telangiectasis&haemorrhages	5	5
Calcification	3	3
Adhesions	2	2
Total	100	100%



Fig. 1: Hydatid cysts of various sizes embedded in liver tissue.

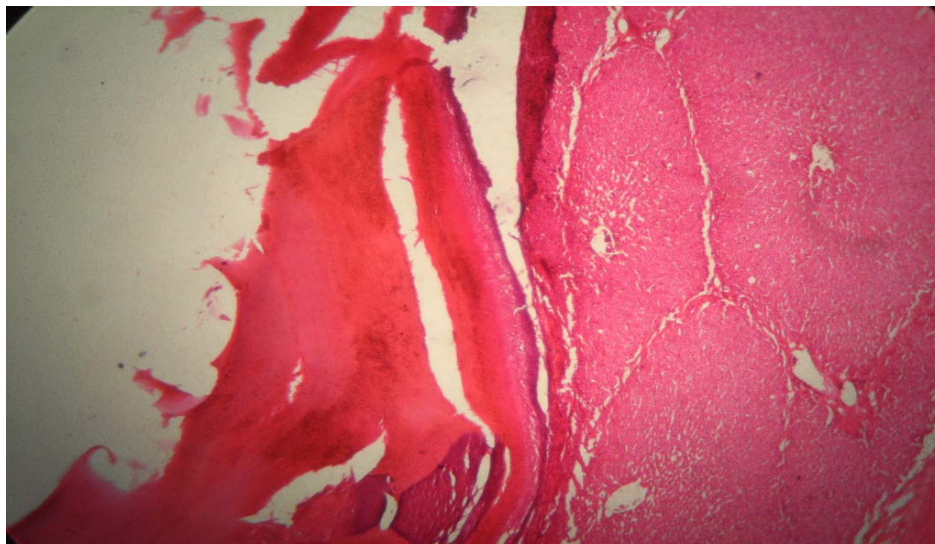


Fig.2: Hydatid cyst in the liver. Note thick cyst wall and pericystic hepatic tissue showing dilated sinusoids with atrophied hepatocytes. (H&E stain, X 40).

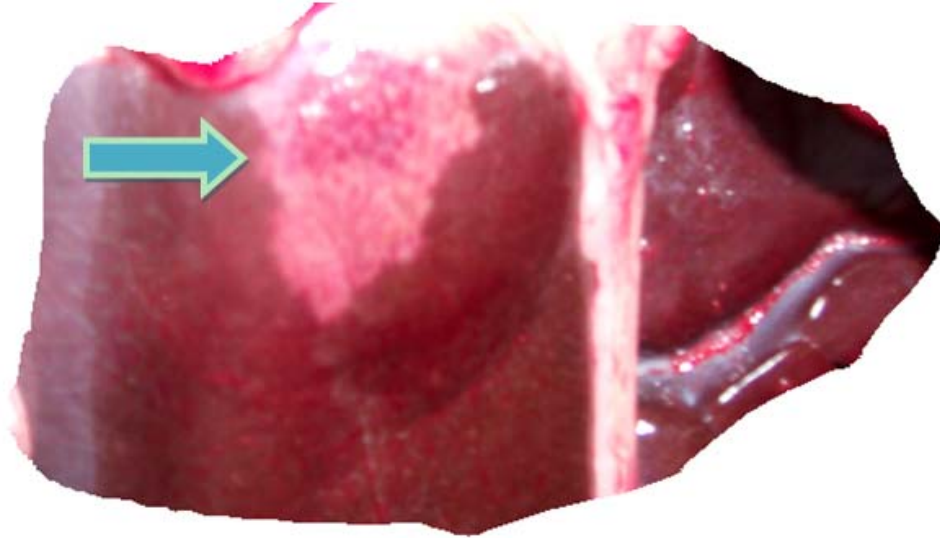


Fig. 3: A liver showing pale necrotic area. (coagulative necrosis).

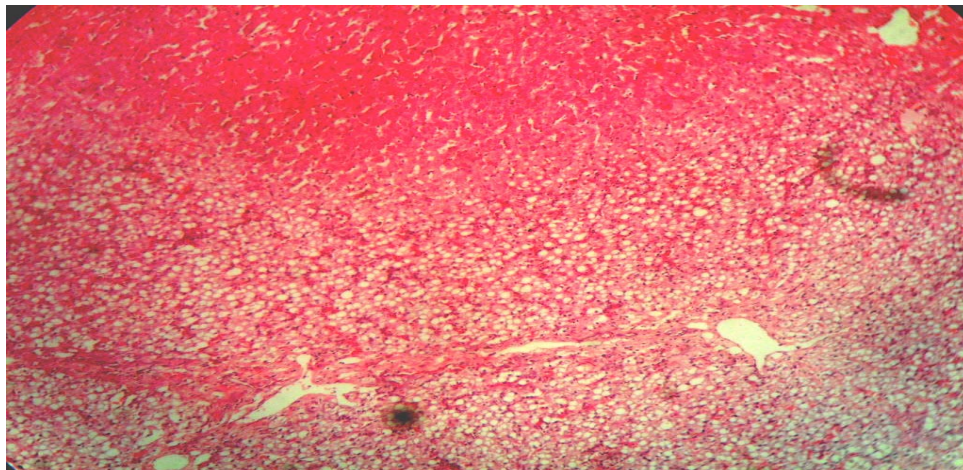


Fig.4: A liver showing severe fatty changes and coagulative necrosis. (H&E stain, X40).

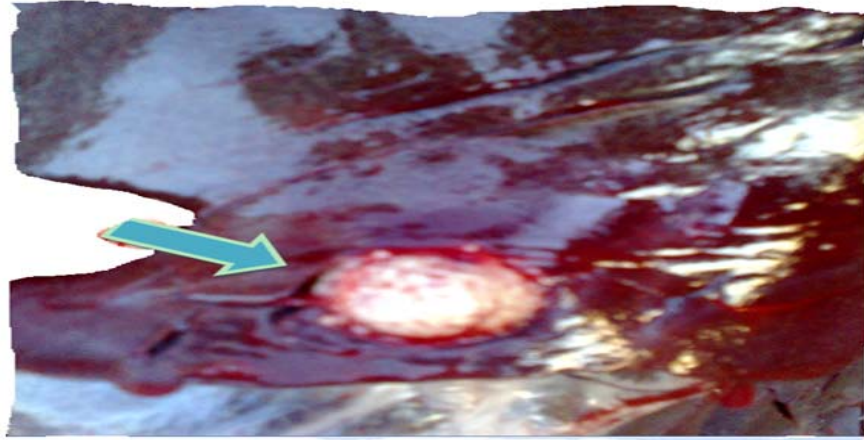


Fig. 5: A liver abscessation. The abscess is bulging from the surface .Notice some degree of congestion in the liver tissue.

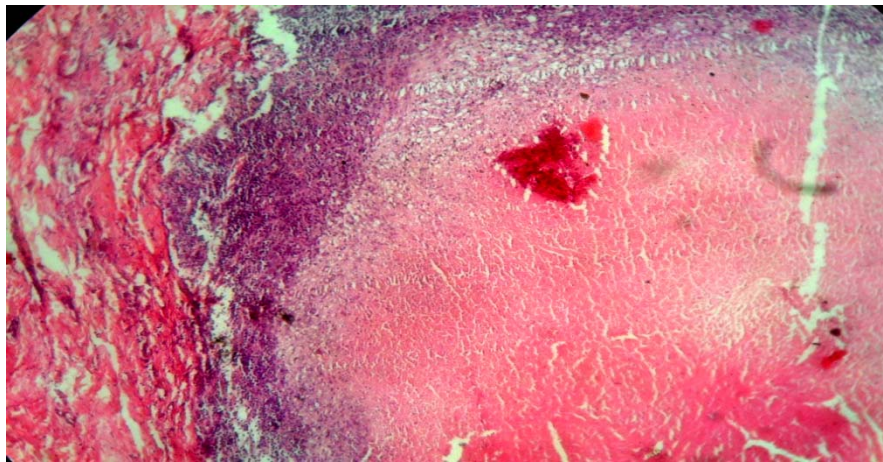


Fig.6: A liver abscessation.Note part of large abscess, necrotic centre. fibrous tissue capsule, with cellular infiltration mainly neutrophils,lymphocytes,and macrophages and proliferation of fibroblast and calcification. (H&E stain, X40).



Fig. 7: Note dark red, enlarged and friable congested liver.

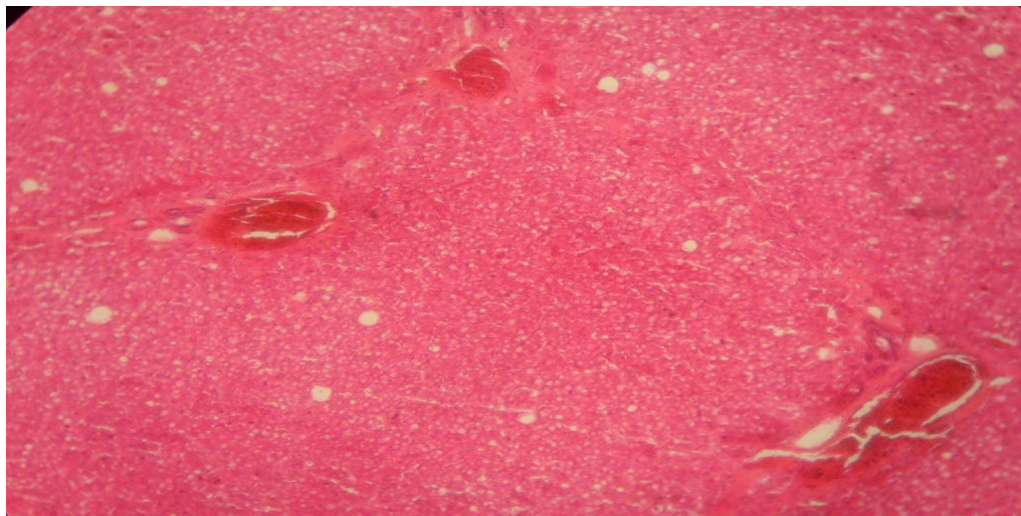


Fig.8: A liver showing congestion .Note distended portal veins with RBCs. (H&E stain, X40).

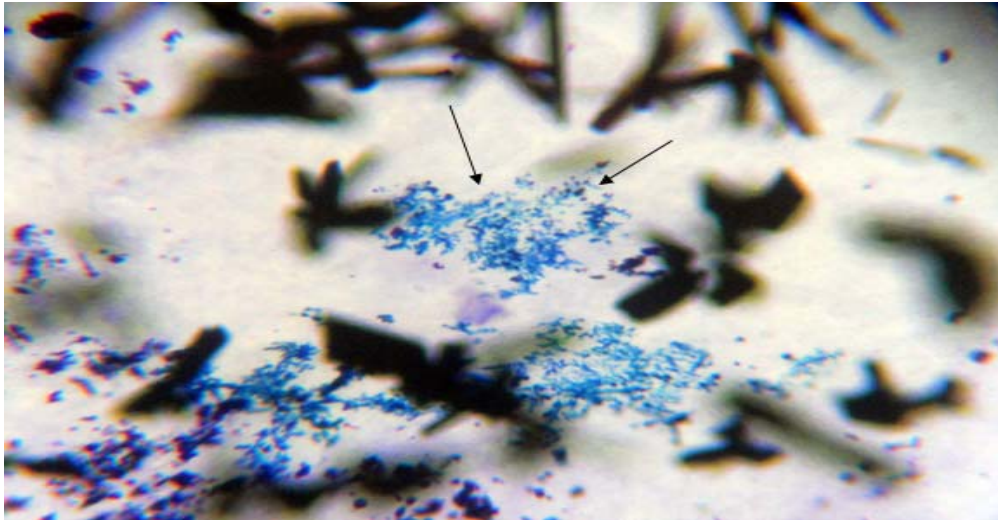


Fig.9: Note presence of gram positive cocci of bacteria in section of liver's abscesses. (Gram's stain X400).

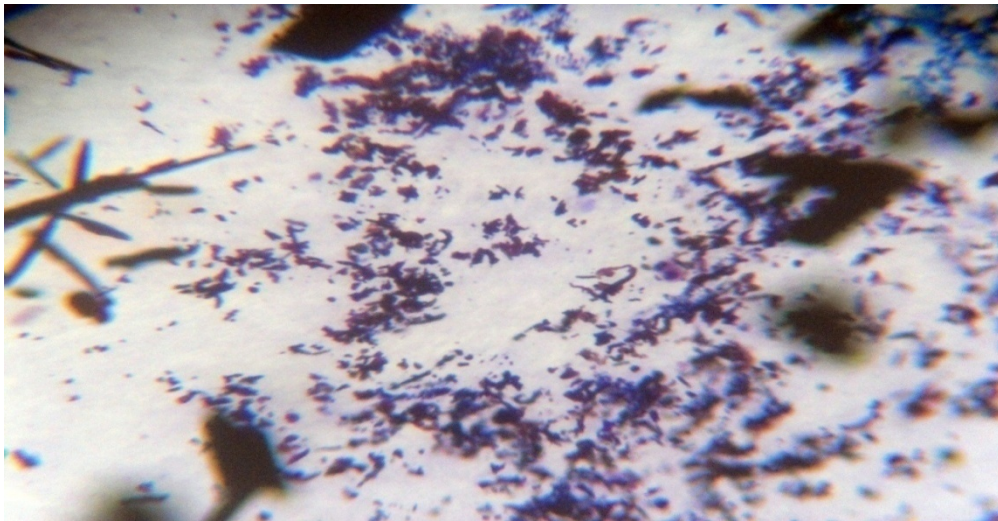


Fig.10: Note presence of gram positive bacilli in section of liver's abscesses. (Gram's stain, X1000).

Discussion

The major cause of liver condemnation in this study was found to be hydatid cysts (33% of the examined samples). These results are similar to the findings of Abdel Rahman (1996) who reported that hydatid cysts caused the highest percentages of condemnation (39.2%) as compared with other causes, in Kordofan State. The results also agree partially with different investigations in other countries who have reported the incidence of hydatid cysts in camel's livers as: 25% in Ethiopia (Muskin Salih.,2011) and 16.5% in Central Somalia (Moallin, 2009). Among ruminants, camels host the highest prevalence rates of hydatid cyst infection followed by sheep, cattle and goats (El Khawad *et al.*, 1979). This could be attributed to the fact that camel owners usually keep dogs with their herds. In this study cirrhosis ranks second as a cause of liver condemnation(15%).This appears similar to that reported by Abdel Rahman (1996) in Kordofan state (14.6%). Shaymaa and Azza (2011) in Egypt reported 12 cases out of 92 camel's livers (13%) having hepatitis and cirrhosis.

In this study the prevalence of necrosis was 13% and this agree partially with Shaymaa and Azza (2011) who reported 8 cases of hepatic necrosis (18.1%). The prevalence of abscesses in this investigation was 9%. This finding is in agreement with that of Abdel Rahman (1996) who reported 10.1%.

Gram's stain for demonstration of bacteria in sections revealed presence of gram positive colonies of bacteria in some sections which showed abscesses by H&E. The results of the present study indicated that 7% of camel's livers were condemned due to fatty changes. This finding is more or less consistent with that of Abdel Rahman (1996) who reported 5.6% and agree partially with Tejsingh *etal.* (2006) who reported 2.91%, also Brad Smith *etal.*(1998) reported hepatic lipidosis in the Camelid. However, contrary to that Shaymaa and Azza (2011) found (47.7%).

In this study congestion was found to be 13% while telangiectasis&haemorrhages were found to be 5% in contrast to Abdel Rahman (1996) who found congestion 3.4%. In this study calcification was

found to be 3% of the examined samples. This appears to be similar to the result of Abdel Rahman (1996) who reported 4.45%.

Conclusions

Many causes accounted for camel's livers condemnations in Tamboul slaughterhouse. Hydatid cysts were the main causative agent leading to liver condemnation. The research defined and elucidated the pathological changes of camel's livers condemned in Tamboul abattoir.

References

- Abdel-Rahman, A.I. (1996)** The aetiology of camel liver poisoning of man in Kordofan States, P.h.D, University of Khartoum.
- Bancroft, J. D., Stevens, A and Turner, D. R. (1996).** *Theory and practice of histological techniques*. Fourth edition, Churchill Livingstone, Edinburgh, London, Melbourne.
- Brad Smith, Susan Tornquist, and Robert Van Saun. (1998).** Hepatic Lipidosis in the Camelid: A Different Perspective, The Ohio State University, Columbus, Ohio.
- Dorman, A.E., 1986.** Aspects of the husbandry and management of the genus *Camelas*. In: A.J. Higgins, (ed.), *The Camel in Health and Disease*, pp: 3–20. Baillier Tindall, London.
- El Khawad, S.E.; Eisa, A.M.; Slepnev, N.K. and Saad, M.B.A. (1979).** *Bull. Anim. Hlth. Prod. Afr.* 27: 249-251.
- FAO–OIE–WHO, 1992.** Animal Health Year Book. *Food & Agric. Org.*, United Nations, Rome, Italy, 46: 206–8.
- Faye B, Abdelhadi O M A, Ahmed A I and Bakheit S A. (2011).** Camel in Sudan: future prospects, *Livestock Research for Rural Development* 23 (10) *Published 10 October 2011*.
- Moallin ASM. (2009).** Observations on diseases of the dromedary in Central Somalia, amkutub@hotmail.com.
- Muskin Salih, Hailu Degefu and Moti Yohannes. (2011).** Infection rates, cyst fertility and larval viability of hydatid disease in camels, *Global Veterinaria*; 7(6) :pp518-522.

- Pratt, P.W., (1992).** Laboratory Procedures for Veterinary Technicians. 2nd ed. American Veterinary Publications, Inc California, pp: 111-116.
- RAB and Wallington EA. (1980).** Carleton's Histological Technique, Fifth Edition; Oxford Medical Publications.
- Shaymaa I. Salem and Azza H.M. Hassan,(2011).** Clinicopathological, cytological and histopathological studies on liver and kidney affections in camels, Global Veterinaria 7 (6): 557-571.
- Tejsingh, G.D., A. Sharms, R.D. Singh and S. Surender, 2006.** Incidence and Pathology of degenerative changes in liver of camels. Veterinary Practitioner, 7(1): 35-36.
- VanSaun, R.J., B. Callihan and S.J. Tornquist, (2000).** Nutritional support for treatment of hepatic lipidosis in a llama. Journal of American Veterinary Medical Association, 217(10): 1531-1535.