Pathological studies on parasitic pneumonia in slaughtered camels infected with hydatid cysts

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Abstract
Hydatidosis is one of the causative agents of parasitic pneumonia in camels. In the present study, lungs of 300 slaughtered camels in the main Cairo abattoir were examined for hydatid cysts. The number and diameter of cysts were recorded and the contents of the cysts were examined microscopically to detect the protoscolices. The results indicated that 83 out of 300 camels (27.67%) were positive to hydatidosis.The fertility rate was 73.2% where 68.3% of fertile cysts were viable. Gross and histopathological lesions in the lungs were identified. Sodium Dodecyl Sulfate-Polyacrylamide Gel Electrophoresis (SDS-PAGE) revealed two distinct patterns for each of hydatid fluid and germinal layer of the cyst suggesting the existence of two strains which were camel and sheep strain. Histopathologically, hydatid cysts wall was composed of three layers, inner germinal, intermediate acellular laminated and outer fibrous connective tissue infiltrated with polymorphnuclear inflammatory cells. These cysts bring about pressure atrophy on the adjacent pulmonary tissues. Many free necrotic and partially evaginated protoscolices were observed in the pulmonary tissues as well as in the lumen of the bronchioles. Meanwhile, some cases were sterile where, no scolices or protoscolices were seen within the cyst. The adjacent pulmonary tissue revealed various degrees of inflammatory reactions including fibrous connective tissue proliferation, necrosis, calcification and inflammatory cell aggregations. Public health problem of zoonotic echinococcosis and the need of preventive measures were discussed.

Key words: *Hydatidosis, Gel Electrophoresis, evaginated protoscolices, SDS-PAGE.*

Introduction

Camels were, and still valuable for riding, baggage, draught, hair hides and as well as meat providers in the arid areas (**Sweet, 1965**). Camel is comparatively endurable and less susceptible to many of the diseases that affect other livestock species in the same area **(Dirie and Abdurahman, 2003)**. However, it is apparent that we know little about the diseases which it suffers. In fact lung infections especially pneumonia are major diseases of domestic animals. Some workers studied camel pneumonia in Egypt and other parts of the world (**Pavlik *et al*., 2002 and Nasr, 2003**).Their studies stated the seasonality in the occurrence of camel pneumonia and mentioned the etiological agents responsible for the condition. Pulmonary diseases are among the emerging problems of camels that are causing considerable loss in production and death (**Bekele, 1999**). Economic losses result from; condemnation of infected carcasses, incapacitation of infected people and from expensive preventive and eradicative programs (**Cleon, 1988**).

The main causes of lung condemnation in slaughter houses were found to be zoonotic parasites (hydatidosis). Several studies indicated that hydatid disease is an endemic in Egypt affecting both humans and domestic animals (**Rahman *et al*., 1992 and Azab *et al*., 2004**). The work in the veterinary field showed that camels are the most infested (36%), although other species are also affected: cattle (4%) and small ruminants (5%). The prevalence rate of hydatid disease is about 14% in dogs and 9% in jackals (**Salem *et al*., 1999**). The most dominant form of hydatidosis in camels is the pulmonary form with a high fertility rate of the cysts. This is in contrast with cattle and small ruminants where, the liver form is the most prominent with less fertility rate of the cysts (**Pangui and Ould, 1996**). In general, single or

multiple cysts are found in the liver and/or lungs and occasionally other organs in different animals. The fully grown cyst is filled with fluid and contains hundreds of protoscolices (**Cleon, 1988**).

Pathogenic effects of hydatid cysts include pressure atrophy of surrounding organs and allergic reactions to hydatid fluid leaks. Pulmonary hydatid cysts may rupture into a bronchus, the contents coughed up and the lesion healed. Hydatid cyst that remain intact eventually die and degenerated but the course is protracted (**Dwight and Randy, 1999).**

Over the last decade diagnosis of hydatid disease in human being was improved due to the use of imaging techniques including ultrasonography, computed tomography (CT scanning) and magnetic resonance imaging (MRI) supported by immunological assays for confirmation of clinical diagnosis (**WHO, 2003**).

Therefore, this study was conducted to study certain aspects of hydatid disease in camels such as incidence, pathological lesions, fertility of cysts, their location, histological structure and viability of protoscolices. In addition, strain identification using gel electrophoresis to obtain more accurate data on the nature of the disease in camels in Egypt.

Materials and Methods

Collection of samples:-

A total of 300 camels slaughtered in the main Cairo abattoir were examined for lung hydatidosis and the degree of involvement in the organ. The detected cysts were removed carefully with the surrounding tissues and transferred in clean plastic bags for further laboratory parasitological and pathological investigations.

Laboratory investigations:-

A- Parasitological Examination:-

1-Morphology:-

Each cyst was measured using divided ruler for determining of the size and its diameter. The wall structure was examined histologically.

2- Hydatid cyst fluid (HCF):

The fluids of fresh cysts were withdrawn using sterile plastic syringes, then centrifuged at 1500 rpm for 30 minutes. The supernatant fluid was collected and stored at -20°C for electrophoretic analysis. The sediment was examined microscopically for detection of fertility. Viability of protoscolices was detected by using 0.1% aqueous eosin, as dead protoscolices take red stain while viable ones remain none stained **(Moazeni and Larki, 2010).**

3-Germinal layer (GL):

The inner layer of each fertile cyst was removed and suspended in Phosphate Buffer Saline (PBS), homogenized, sonicated and stored at -20°C for electrophoretic analysis.

4- Electrophoretic analysis of HCF and GL:

Supernatant fluid of HCF and GL of 25 camel hydatid cysts were electrophoresed in Genetic Engineering Unite, Agricultural Research Center using 10% Sodium Dodecyl Sulfate-Polyacrylamide Gel (SDS-PAGE) under reducing conditions according to Laemmli (1970). The molecular weights of the parasite proteins were estimated by comparing their electrophoretic motilities with standard molecular weight marker (SIGMA) after electrophoresis in the same gel.

B- Histopathological examination:-

Selected portions from camel lungs including cysts (after aspiration of the fluid from the cysts) were fixed in 10% neutral buffered formalin solution for 24 hours and routinely processed by the standard paraffin embedding technique. Tissue sections were cut at 3-4 microns and stained with haematoxylin and eosin stain according to **Bancroft *et al*. (1994)**.

Results

A- Parasitological studies:-

The prevalence of hydatidosis in the examined camels slaughtered in Cairo abattoir was 83 out of 300 (27.67%), and the total number of detected cysts was 224 with average of 2.7 cysts per camel as shown in table 1. The fertile cysts were 164 out of 224 (73.2%), and the viable cysts were 112 out of 164 (68.3%), as shown in Table 2.

The indicated cysts were spherical or elongated in shape and vary in size from 10-13 x 7-9 cm in diameter (Fig.1).

The cysts were filled usually with clear to pale

yellow fluid containing different stages of microscopical scolices (non evaginated and evaginated measuring 85-110 µ and 215-260 µ in length respectively) as shown in fig. 2 a, b and c. Some cysts were very small, ill developed and contained no scolices. Cysts containing scolices were considered fertile, where those free from scolices were sterile. Some rostellar hooks were found in HCF, measuring 18-22 µ length (fig. 2d).

The wall of the cyst consists of an outer thick cuticle, concentrically laminated with an inner germinal layer. Small vesicles were attached to the inner layer (fig. 3).

SDS-PAGE revealed two patterns for each of hydatid cyst fluid (HCF) and the germinal layer (GL) (fig. 4). The quantitative analysis revealed 4 bands in both HCF and GL in the first pattern or lane-1, whereas, the second pattern or lane-2 revealed 5 bands in HCF and only 3 bands in GL expressed as molecular weight (MW) values as shown in table 3 and rate of flow (RF) values in table 4.

The first electrophoretic pattern showed 17 fertile cysts out of 19 (89.5%) and 2 sterile cysts (10.5%), where the second pattern showed only one fertile cyst out of 6(16.7%) and 5 sterile cysts (83.3%) with total of 18 fertile cysts out of examined 25 cysts (72%) and 7 sterile (28%) as shown in table 5.

B- Pathological studies:-

Macroscopical examination:

Single and multiple cysts of varying sizes were seen either embedded or protruded over the surface of the lung. In cut sections of the cyst, white friable layers of the cyst wall were separated; only rigid fibrous layer capsule were attached to the lung tissues. The adjacent lung tissues were congested with streaks of hemorrhages with adhesion of pleura. In two lungs, white firm abscesses (1-2 cm in diameter) appeared on the parenchymal surface and on cut section, caseous material oozed out.

Microscopical examination:-

There was more than one fertile cyst detected in the lung parenchyma. The hydatid cyst wall composed of three layers, the outer layer (adventitial layer) or ectocyst (f), is a fibrous capsule produced by the host, the intermediate layer or pericyst is acellular laminated (Im) hyaline cuticular membrane followed by the inner layer or endocyst is one-cell-membrane attached to its interior surface known as germinal membrane (gm). The adventitial layer revealed focal and diffuse leukocytic infiltration (Fig. 5a & b). The germinal layer proliferated into the lumen to form free vesicular structures known as hydatid sand (Fig. 6a & b). In few instances, daughter cysts were seen under the laminated layer (Fig. 7a & b). In some cases, many free necrotic and partially evaginated protoscolices with branched tracts were observed in the pulmonary tissues as well as in the lumen of the bronchioles with congestion of the perialveolar capillaries (Fig. 8 a & b and Fig. 9 a & b). Meanwhile, no protoscolices were detected. The adjacent lung parenchyma was atelectatic and compensated by areas of emphysema (Fig.10a) with perivascular oedema and peri-bronchial mononuclear cell infiltrations (Fig. 10b). Atrophy of the neighouring pulmonary tissue was detected. Haemorrahges were also observed in the interstitial pulmonary tissues (Fig.11). Some alveolar lumina revealed various degrees of fibrin as well as cellular exudates where fibrinous exudate consisted of fibrillar network of fibrin admixed with mononuclear and polymorphonuclear cells (Fig 12). Moreover, the bronchiolar epithelium appeared hyperplastic which form papillary projections into the lumina, while the bronchial mucosa was oedematous and infiltrated with mononuclear cells (Fig. 13 a & b).

Furthermore, there were increased fibrous connective tissue proliferations in the pleura which extended to invade large parts of the pulmonary tissue together with diffuse and multifocal inflammatory cell aggregations (Fig. 14a & b). Also, neovascularization with thick walled blood vessels were evident in the subpleural pulmonary tissue (Fig. 15). Large subpleural abscess were recognized in 2 cases, which consisted of central area of liquifactive necrosis, few calcification and mixed populations of in-

flammatory cells which consisted principally of intact and damaged neutrophils, histocytes and lymphocytes with inconspicuous outer fibrous capsule was observed (Fig.16). In two cases, one old cyst in the lung was seen represented by focal eosinophilic structurless necrotic zone within the adventitial layer which showed focal calcification at the periphery and surrounded by leukocytic inflammatory cells infiltrate while, the adjacent laminated layer appeared fragmented (Fig.17). In one case, multichambered cyst was seen due to penetration of the cyst by the fibrous tissue through adventitial layer and surrounded by leukocytic inflammatory cell infiltrations (18 a & b).

Table (1) . The prevalence of hydatidosis in camels slaughtered in main Cairo abattoir.

Examined animals	Infected animals	(%)	Total Number of cysts	Average (cysts/ animal)
300	83	27.67%	224	2.7

Table (2). The prevalence of fertility and viability of camel hydatid cysts.

Number of cysts	Fertile cysts	(%)	Viable cysts	(%)
224	164	73.2%	112	68.3%

Table (3). Molecular weight values (SDS-PAGE) of hydatid fluids and germinal layer collected from camels infected with cystic echinococcosis.

M.W. Values	Hydatid cyst fluid		Germinal layer	
	Lane 1	Lane 2	Lane 1	Lane 2
Band 1	364.783	359.565	364.783	364.783
Band 2			312.609	
Band 3	260.435	265.652		
Band 4		149.207		
Band 5	49.683	55.58	53.314	52.77
Band 6	35.5	38.475	37.607	38.475

Table (4). Rate of flow values (SDS-PAGE) of hydatid fluids and germinal layer collected from camels infected with cystic echinococcosis.

R.F. values	Hydatid cyst fluid		Germinal layer	
	Lane 1	Lane 2	Lane 1	Lane 2
Band 1	0.117	0.121	0.117	0.117
Band 2			0.158	
Band 3	0.198	0.194		
Band 4		0.279		
Band 5	0.583	0.538	0.555	0.559
Band 6	0.745	0.7	0.713	0.7

Table (5). The prevalence of SDS-PAGE electrophoresis and fertility of camel hydatid cysts.

Patterns	SDS-PAGE	(%)	Fertile cysts	(%)	Sterile cysts	(%)
Pattern.1	19	76%	17	89.5%	2	10.5%
Pattern.2	6	24%	1	16.7%	5	83.3%
Total	25	100%	18	72%	7	28%

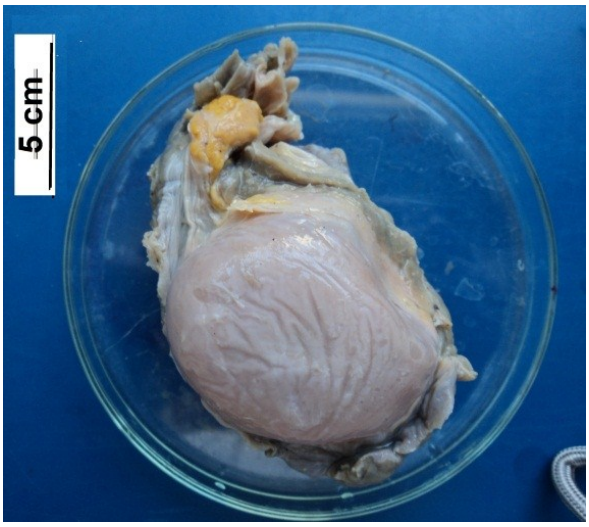

Fig. (1). Hydatid cyst from lung of camel.

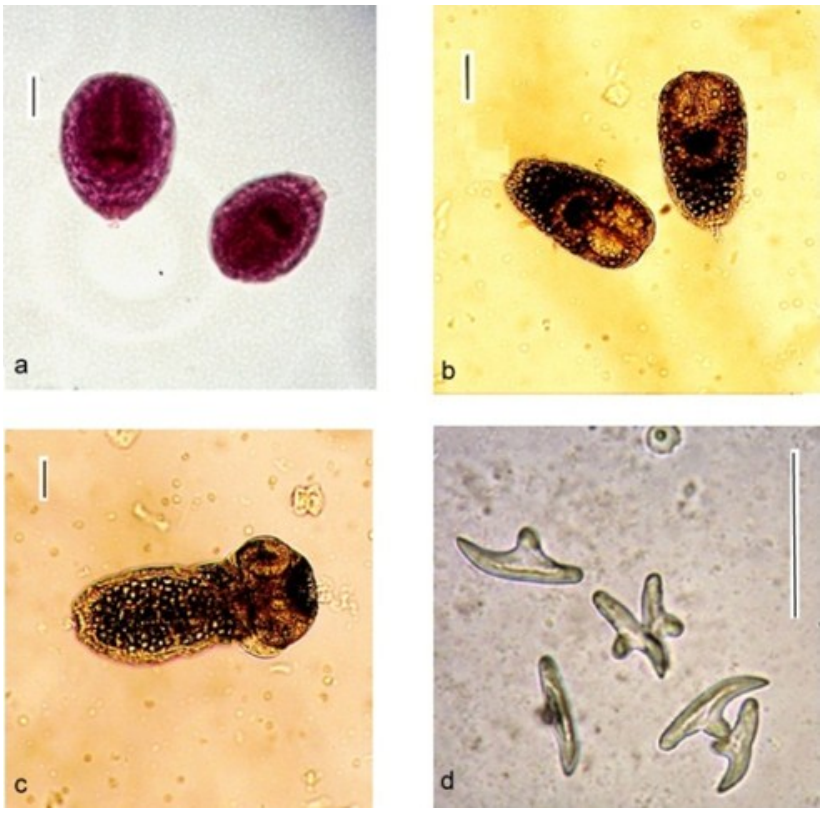

Fig. (2). Different stages of microscopical scolices, a,b-Non-evaginated scolices. c-Evaginated scolex. d- Rostellar hooks (Bar=25µ).

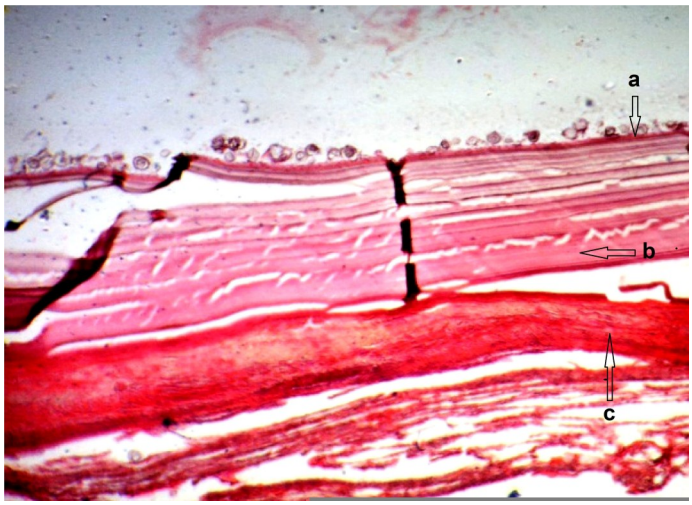

Fig. (3). The wall of hydatid cyst showing, a) Inner germinal layer from which small vesicles were attached. b) Concentrically laminated layer. c) Outer thick cuticle (x 100).

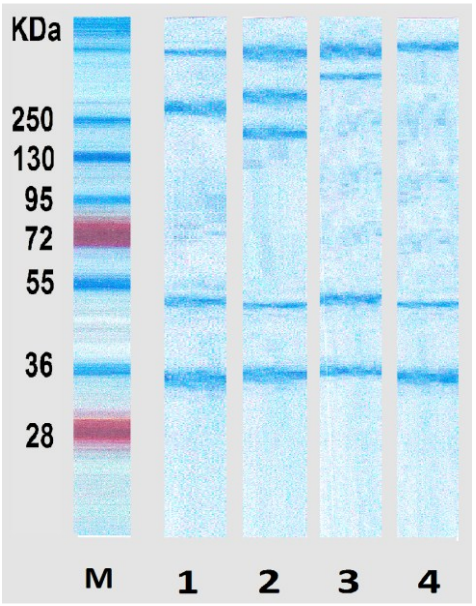

Fig. (4). SDS-PAGE patterns of camel hydatid cyst protein. (M=Marker), 1=HCF, pattern-1; 2=HCF, pattern-2; 3=GL, pattern-1; 4= GL, pattern-2.

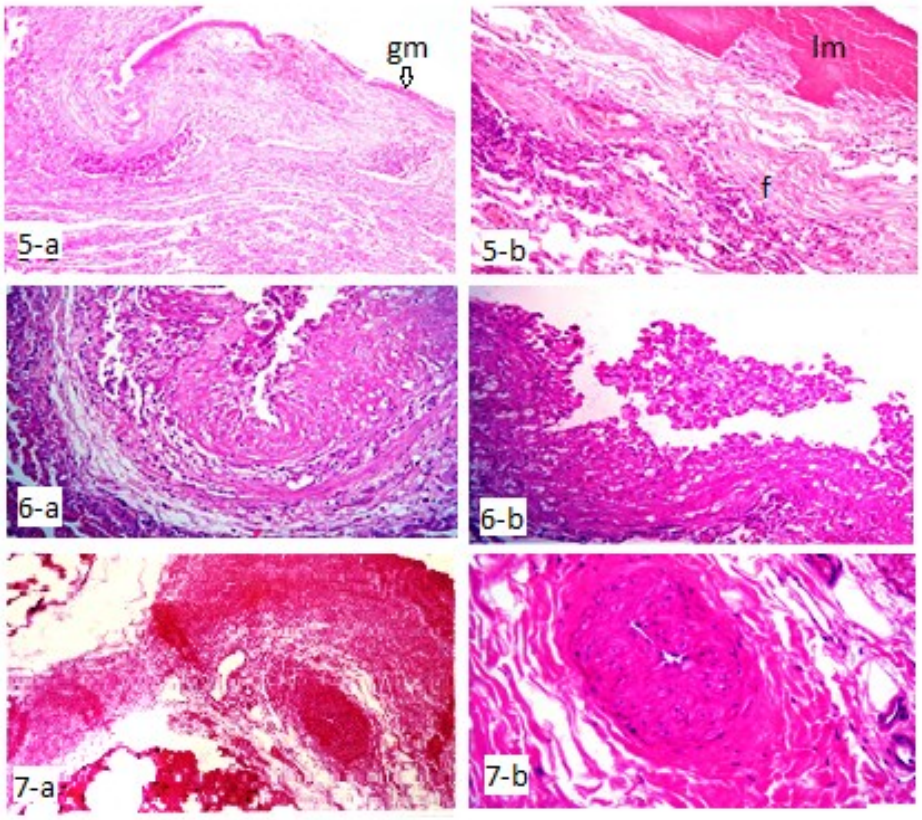

Fig. 5 (a & b): Lung of camel illustrating laminated layer of the cyst (1m) (pericyst) and the germinal layer (gm) (endocyst), the two layers were surrounded by fibrous connective tissue capsule (ectocyst) (f) which showed focal and diffuse leukocystic infiltration. (H & E Stain, a: X 200 & b: X 400).

Fig. 6 (a & b): Lung of camel depicating a) proliferation of the germinal layer of the hydatid cyst into its lumen. b) hydatid sand in the lumen of the cyst (H&E Stain, a: X 200 & b: X 400).

Fig. 7 (a & b): Lung of camel showing daughter cyst a) H & E stain X 100. b) Higher magnification of a) (H & E stain X 400)

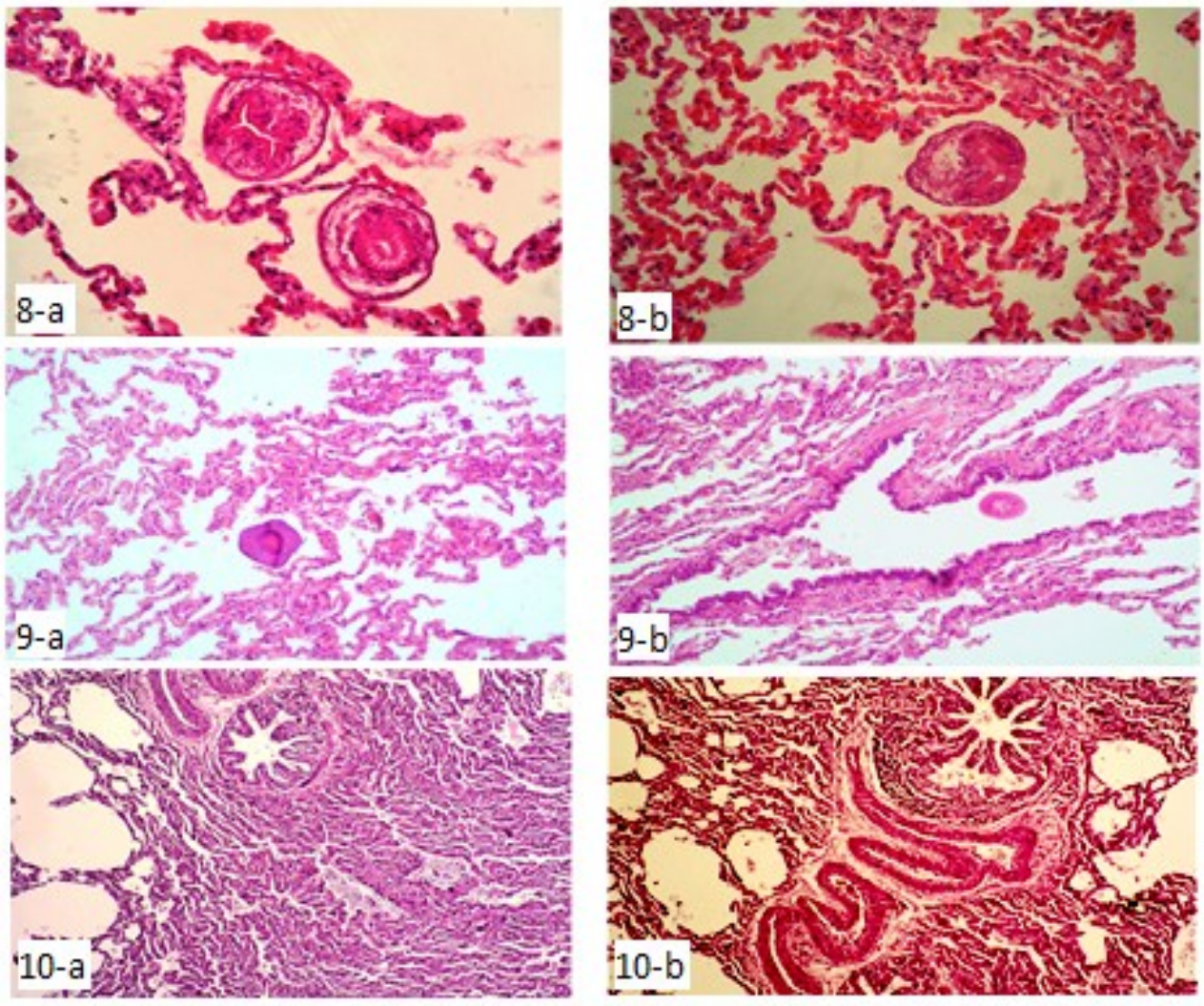

Fig. 8(a& b): Lung of camel showing evaginated protoscolices with branched tracts. The adjacent pulmonary tissue showed congestion of the perialveolar capillaries. (H & E Stain X 400).
Fig. 9(a& b): Lung of camel showing necrotic scolex a: in the pulmonary tissue and b: in the bronchiole. (H &E Stain, a: X 200& b: X 100).
Fig. 10(a): Lung of camel showing atelectasis of the pulmonary tissue and compensated areas of emphysema (H & E X 100).
Fig. 10(b): Lung of camel showing perivascular oedema and peribronchial mononuclear cell infiltrations and emphysema (H&E stain x: 100).

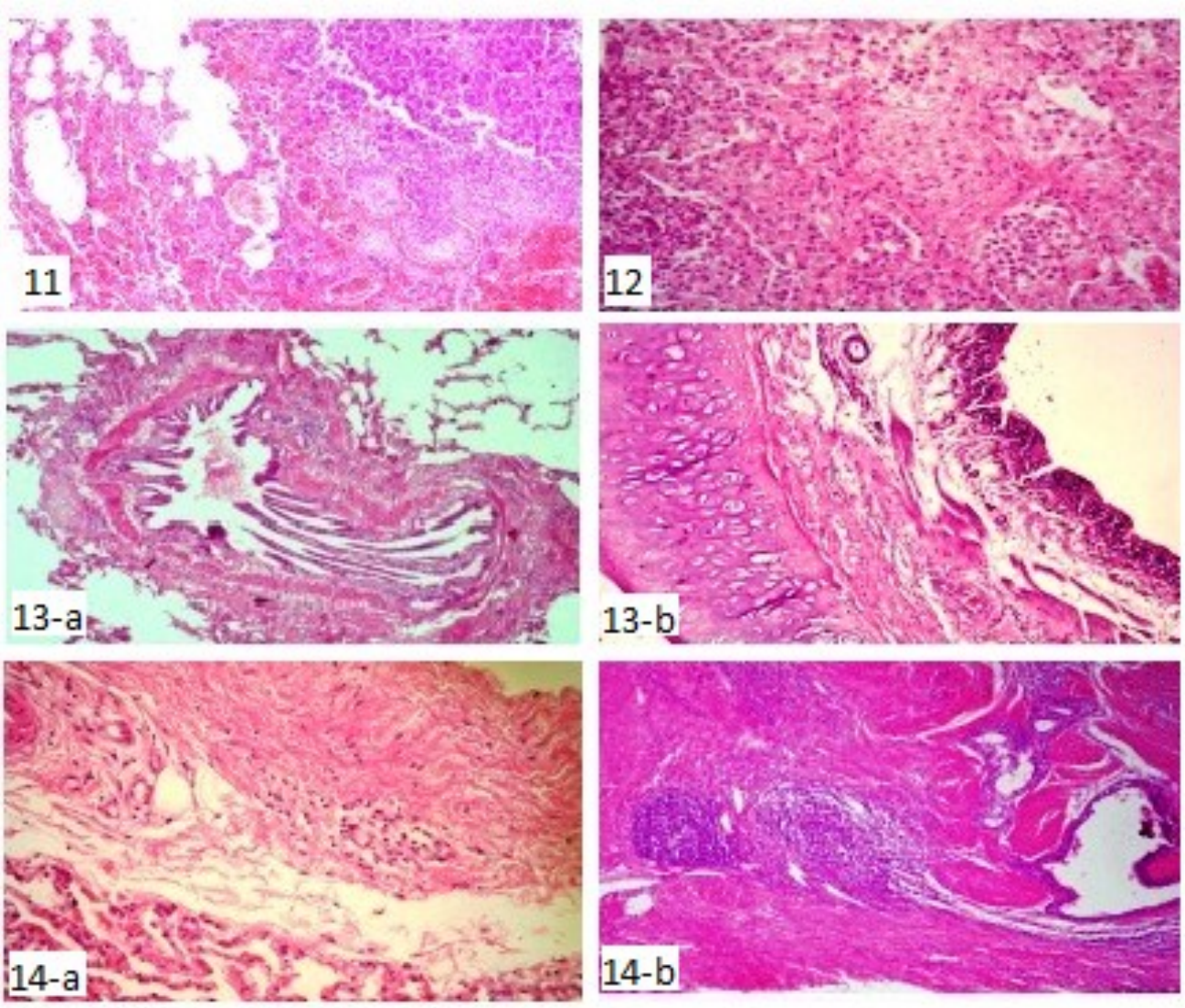

Fig. 11: Lung of camel showing hemorrhages in the interstitial pulmonary tissue, moreover, the alveolar lumins were filled with cellular exudates (H & E Stain X 200).

Fig. 12: Lung of camel showing fibrinous as well as cellular exudate within the alveolar lumina. The fibrinous exudate consisted of pink fibrillar network of fibrin admixed with mononuclear and polymorphonuclear leukocytic cellular exudates (H & E Stain X 400).

Fig. 13(a): Lung of camel showing hyperplastic proliferation of the bronchial epithelium which forms papillary projections toward the lumina. (H & E Stain X 200). Fig. 13(b): Lung of camel showing oedema of the bronchial mucosa admixed with few mononuclear cells (H & E Stain X 400).

Fig. 14(a& b): Lung of camel showing increased fibrous connective tissue proliferation with a) diffuse inflammatory cells infiltrations in the pleura. b) focal mononuclear cell infiltrations in the pulmonary tissues. (H & E Stain a: X 400 & b: X 200).

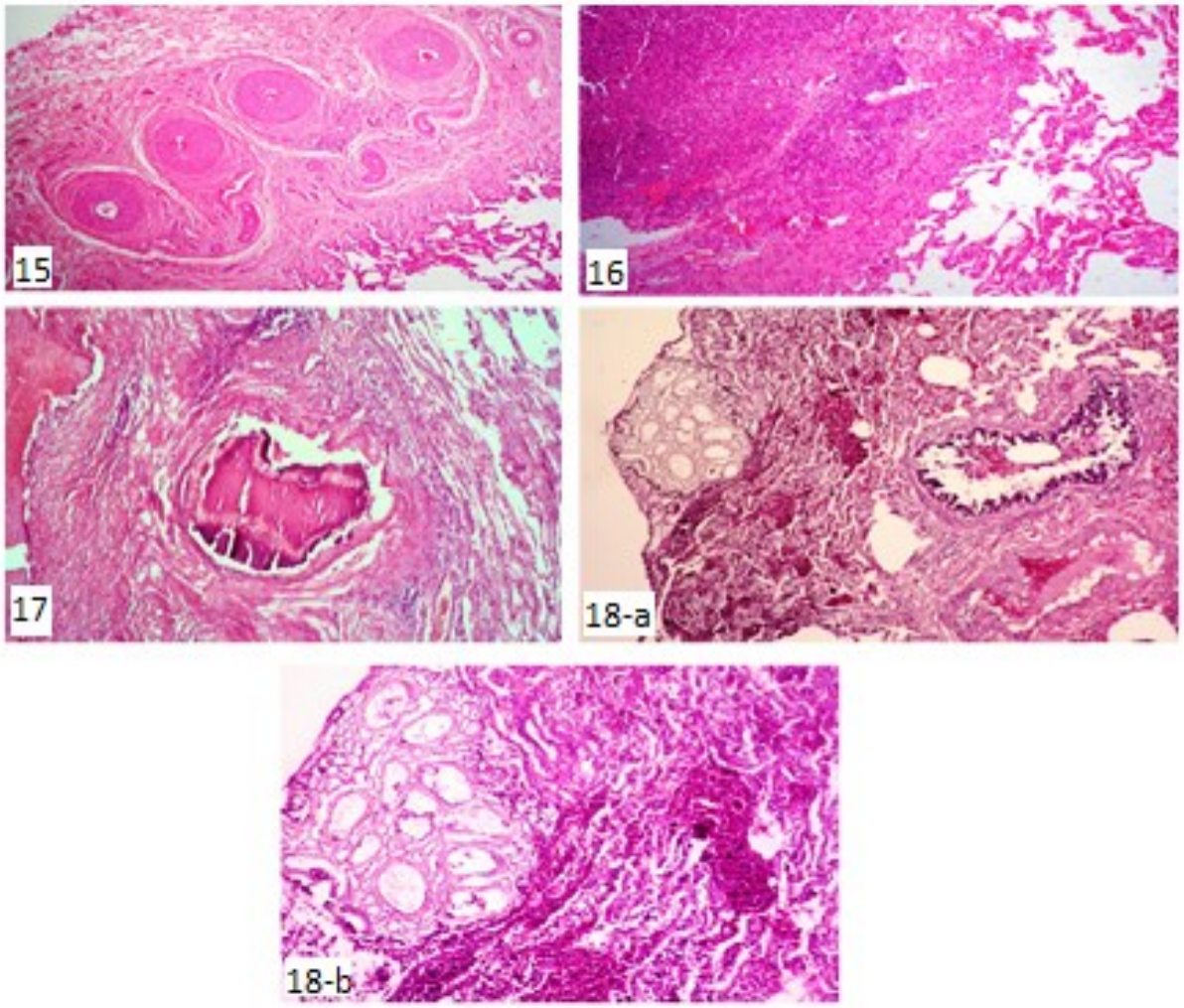

Fig. 15: Lung of camel showing newly formed blood vessels. There were fibrous connective tissue proliferation which formed concentric lamellaticus around the newly formed blood vessels (H & E Stain X 200).

Fig. 16: Lung of camel showing pulmonary abscess (H. & E. Stain X 100).

Fig. 17: Lung of camel showing old cyst consisting of eosinophilic patch of focal necrosis which undergo calcification and surrounded by leukocytic inflammatory cells. (H & E Stain X 400).

Fig. 18(a& b): Lung of camel showing multichambered cyst surrounded by inflammatory cell infiltration (H & E Stain, a: X 100 & b: X 200).

Discussion

Cystic echiniococcosis is one of the major cosmopolitan parasitic zoonoses in the Middle East and North Africa from Morocco to Egypt. It is caused by *E. granulosus* which is a parasite of dogs and some other canids as the definitive host. Its larval stage is a hydatid cyst which is acquired by camel as well as other animals including man. While sheep and goats appear to be the most common domestic intermediate host, the epidemiological data strongly suggest that camels are the reservoir of infection for human (**Macpherson *et al*., 1986**).

In the present study, the prevalence of hydatidosis in camels was 27.67% and the overall number of cyst detected in 83 infected animals was 224 cysts (average 2.7 cysts per animal). Nearly similar results were previously recorded in Egypt, 25.83% (**El-Badr *et al*., 2009**), 26.5% (**Ahmed, 1990**), 31% (**Rahman *et al*.,**

1992), in Ethiopia, 30.8% (**Bekele, 2008**) and in Mauritania, 30.1% (**Salem *et al*., 2011**). In contrast, our result is low compared to the prevalence rates of high endemicity areas in Sudan 54.4% (**Saad *et al*., 1983**), and 55.5% in Nigeria (**Dada and Belino, 1978**).The difference in the prevalence rates may be due to rearing of animals in different localities, and the higher prevalence may be attributed to the close association of such animal with wild carnivores living mostly in the desert. Moreover, the improper hygienic measures in the abattoirs give chance to stray dogs which consume infected offal's and lead to spread of hydatidosis among animals.

Concerning fertility and viability in the present study, it was found that 73.2% of detected cysts were fertile and 68.3% of fertile cysts were viable. These results were roughly in accordance with those reported by **Al-Rashed *et al*. (1994)** and **El-Badr *et al*. (2009).** The degree of infection help to indicate the risk for human infection via dogs, but cyst fertility rates determine the real role of camel as food animal in the cycle of infection (**Gusbi *et al*., 1990**). Moreover, high fertility rates increase the risk of secondary peritoneal hydatidosis as a serious complication due to large size cysts, thin wall and intra-thoracic rupture (**Salem *et al*., 2011**).

The morphological features of the isolated cysts and its wall structure which was characterized by thick cuticle, laminated, and germinal layer with protoscolices were identical to hydatid cysts as described by **Soulsby (1982).**

The strain characterization is particularly important in regions where more than one species of livestock intermediate host exists and where there is the possibility of different cycles of transmission and sources of infection for humans (**Thompson and Lymbery, 1995).** Clearly biochemical analysis can provide much valuable information on the identification of *E. granulosus* strains from different hosts **(McManus, 1981).** SDS-PAGE revealed two different patterns for each of (HCF) and (GL) expressed as (MW) values and (RF) values**.** The first pattern showed 4 bands in HCF (1,3,5 and 6) expressed as MW and RF values. The second pattern showed nearly the same values of 4 bands with an additional band-4 (1,3,4,5 and 6). Concerning germinal layer, the electrophoretic analysis revealed 4 bands in the first pattern (1,2,5 and 6) where only 3 bands were observed on the second pattern (1,5 and 6). It is clearly found that there were 3 common bands (1,5,6) in the two patterns in both HCF and GL, where band-3 appears only in HCF in both patterns. Moreover, a specific band-2 appears only in the 1st pattern of GL, whereas band-4 is a specific for 2nd pattern in HCF. The first pattern constitutes 76% of the tested cysts with fertility rate 89.5% while the second pattern was 24% with a fertility rate 16.7%. The results of the electrophoretic patterns suggested the existence of two strains of hydatid cysts in the examined camels. In the present study**,** the cysts of 1st pattern were well developed, highly fertile, demonstrated good adaptation to camel as intermediate host and seemed to be of camel strain. On the other hand, the cysts of 2nd pattern showed bad adaptation and lower fertility rates and seemed to be of sheep strain as previously explained by **Salem *et al*. (2011**). Preliminary epidemiological studies on strain specificity of *Echinococcus granulosus* obtained with PCR in Egypt revealed the presence of a camel strain, G6 (**Azab *et al*., 2004)** and sheep strain, G1**(Abd El Baki *et al*., 2009).**The two strains are circulating between sheep, goats, cattle, camels and human **(Sadjjadi, 2006).**

The presence of G1 strain in camels incriminated sheep as a source of cystic echinococcosis for camel. Moreover, molecular characterization of the parasite indicated that human and camel isolates having similar pattern and highest similarity coefficient (**Azab *et al*., 2004**). They concluded that human cases in Egypt are of camel strain; and camel is an important host for the transmission of human infection. These results indicated the serious public health problem of human echinococcosis and the need of strict preventive measures at slaughter places, and early detection and treatment.

Regarding the pathological examination, the macroscopic study revealed single and multiple cysts were seen protruded over the lung surface which were spherical or irregular in shapes, similar results were observed by **Jubb *et al.* (1993)** and **Geeta *et al.* (2010).** Regarding the histopathological examination, the wall of the cyst consisted of an inner germinal (endocyst) and an intermediate hyaline layer of parasitic origin while the reaction of the host to the invading parasite is responsible for the formation of the pericyst which is made up of fibrous connective tissues and surrounded by focal and diffuse leukocytic inflammatory cell infiltrations. These results were in agreement with **Sulata *et al.* (2009)**.

The greater thickness of the germinal membrane suggested greater parasitic activity which may explain more rapid growth rate of the cyst, whereas the thicker adventitial layer of the host act as tissue support of the hydatid cyst and restrict its growth as described by **Barness *et al.* (2011).** The germinal layer was proliferated into vesicular structure in the present study known as hydatid sand as mentioned by **Kattan (1977).** Meanwhile, **Michael *et al.* (1982)** stated that rupture of the scolices released sediment known as hydatid sand. When hydatid sand are liberated into the host tissue, they give rise to secondary hydatid cyst **(Kattan, 1977).**

Daughter cysts were seen in few instances in the present study. **Kattan (1977)** and **Dwight and Randy (1999)** explored out that daughter cysts may be found free in the fluid filled cyst cavity or attached to the germinal membrane (endogenous daughter cyst) or in the pericyst space between the hydatid membrane and the host connective tissue capsule (exogenous daughter cyst). Some daughter cysts may be spread and developed hydatid disease in other parts of the body **(Warnery** and **Ruger, 2002). Also, Kattan (1977)** mentioned that daughter cysts are responsible for clinical signs of "hydatid thrill" felt when percussing over the area of hydatid cyst.

In the present study, many necrotic and partially evaginated protoscolices were seen in the pulmonary tissues as well as in the lumen of the bronchioles. Similar results were observed by **Cleon (1988)** and **Gianlorenzo *et al.* (2007).** Presence of protoscolices indicates fertility of the cyst as mentioned by **Mario *et al.* (2002). Michael *et al.* (1982)** stated that, scolex is the unit of infectious for the definitive host.

In the present study, the adjacent pulmonary tissue revealed various degrees of tissue reactions including haemorrhages, hyperplastic proliferation of the bronchial epithelium, oedema of the bronchial mucosa, perivascular oedema, focal aggregations of mononuclear cells, fibrous connective tissue proliferation in the pulmonary tissue, thick-walled new blood vessels formation. Similar results were observed by **Gianlorezo *et al.* (2007)** and **Fernanda *et al.* (2011).**

Warnery and Ruger (2002) and Geeta *et al.* (2010) mentioned that hydatid diseases were associated with chronic inflammatory reaction. The dynamic equilibrium between parasite growth and acquired immunity in which parasite infection impaired the accessory action of macrophages in lympho-proliferative responses as mentioned by **Dixon (1997).**

Old cysts were found in 2 cases in the present study consisted of eosinophilic structurless necrotic zone with focal calcification and surrounded by leukocytic inflammatory cell infiltrations while the laminated layer appeared fragmented. Similar results were observed by **Warnery** and **Ruger (2002)** and **Barness *et al.* (2011).**

Cystic degeneration occurs spontaneousl undergoing necrosis and calcification **(Thomas *et al.*, 1997).** Meanwhile, **Dwight and Randy (1999)** and **Rogan *et al.* (2006)** stated that cyst degeneration may occur at any point in development. Also, **Barness *et al.* (2011)** mentioned that, leukocytes in the adventitial layer play a greater role in cyst degeneration.

In the present study, sub pleural abscess were recognized with central core of coagulative necrosis and focal calcification. This result indicates bacterial infection and these attributed to impaired body immunity of the animal. Lung abscess and empyema may occur as sec-

ondary bacterial infection in cases of pulmonary hydatid cyst **(Tsieh, 1988).**

In the present study, multichambered cyst developed due to formation of septa by the degenerating adventitial layer and surrounded by inflammatory cell infiltration as mentioned by **Barness *et al*. (2011).** Meanwhile, **Eckert *et al*. (2001)** and **Bortoletti (2007)** mentioned that multivesicular cyst can be developed from numerous daughter cysts of different sizes present in the lumen of the cyst. In spite of a multilocular appearance in all these cases, the cyst is still referred to as unilocular produced by *E. granulosus as* mentioned by **Gutierrez (1990)** and **Barness *et al*. (2011).**

Conclusion:

Camel and sheep strains were identified from hydatid cysts of the infected camels. So, hydatid disease plays an important role in camels which act as a reservoir for human infection and great attention should be directed to camel populations. The control measures of hydatidosis involve destruction of stray dogs and preventing these dogs from access to condemned infected organs in slaughter houses. Hygienic disposal of dead livestock or fresh offal's will control the spread of infection.

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دراســات باثولوجيـــة عـلى الالتهـاب الرئـوي في الجمـال المذبوحـــة المصابة بالحويصـــلات المائيـــة

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الملخص العربـي

لا تزال مشكلة الحويصلات المائية تمثل أهمية صحية عامة في العديد من البلاد وفي مناطق عدة. لذلك تم فحص الأكياس المائية في رئتي 300 من الجمال المذبوحة في مجزر القاهرة. تم تسجيل عدد الأكياس وقطرها وفحصت محتوياتها بالمجهر للكشف عن الطور المعدى. أشارت النتائج إلى أن 83 من أصل 300 من الإبل (27.67٪) كانت إيجابية. وكان معدل خصوبة الحويصلات 73.2٪ ومعدل الحيوية 68.3٪ من الحويصلات الخصبة. تم تحديد التغيرات الباثولوجية والتشريحية المرضية في الرئتين. كشف التحليل الكهربائي للسائل والطبقه الداخليه للحويصلات عن وجود نمطين مميزين مما يدل على وجود سلالتين من الحويصلات التي يبدو أن تكون سلالة الإبل و سلالة الأغنام.

أظهرت الدراسة الباثولوجية ان جدار حويصلة الهيداتيد يتكون من ثلاثة طبقات: طبقة داخلية منبتة وطبقة وسطى رقائقية غير خلوية وطبقة ليفية خارجية بها ارتشاحات من الخلايا الالتهابية. تسبب هذه الحويصلات ضمور للنسيج المجاور لها من الرئة بضغطها عليه. كما أوضحت الدراسة ظهور عديد من رؤوس الدودة الشريطية بعضها مقلوب جزئيا والأخرى متنكرزة بينما توجد بعض الحالات ليس بها هذه الرؤوس (عقيمة) فى نسيج الرئة. كذلك أوضحت الدراسة ان نسيج الرئة المجاور للحويصلات به درجات التهابية مختلفة منها ازدياد النسيج الليفى والتنكرز والتكلس وتجمعات للخلايا الالتهابية.

تمت مناقشة المشكلة الصحية العامة لانتقال المرض للانسان، وضرورة اتخاذ التدابير الوقائية اللازمة.