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Mycoplasmosis in Slaughtered She-Camels: Pathological Studies

[1]Abeer E. El-Metwally, [2]Yassin M.H. and [3]Hanaa A. El-Hallawany

[1,3]Department of Pathology, Animal Reproduction Research Institute (A.R.R.I.), Giza, Egypt
[2]Department of Reproductive Diseases Animal Reproduction Research Institute (A.R.R.I.), Giza, Egypt

Abstract: Mycoplasma is mainly an infectious agent of lung. This work aimed to detect such organism in lung and genital tract of 100 she-camels, slaughtered at Kerdasa Abattoir, Egypt during a period of 14-months (from January 2009 to March 2010). Tissue samples were taken from lung, uterus, cervix, vagina and mammary gland for mycoplasma isolation and histopathological evaluation. The isolation rate of mycoplasmas was 6% from lung and 2% from vagina, no mycoplasma was isolated from uterus, cervix and mammary gland and also no ureaplasmas was detected in all samples. All isolates were identified as *Mycoplasma arginini*. The histopathological lesions in lungs were diffuse suppurative bronchopneumonia and diffuse fibrinosuppurative pleuropneumonia with caseous necrosis. The microscopic examination of uterine tissues revealed chronic endometritis with fibrosis and granulomatus endometritis were noticed in four cases (4%) which were characterized by granulomatus focci, consisted of lymphocytes, histiocytes and multinucleated giant cells situated mostly in the subepithelial layer. The affected cervix and vagina showed chronic granular cervacitis and vaginitis with diffuse and focal lymphocytic and plasma cells infiltrations at the subepithelial region. The Mammary glands revealed acute diffuse purulent mastitis associated with galactophoritis in two cases and chronic mastitis which was characterized by chronic galactopheritis with extensive alveolar atrophy were observed in other cases. Mycoplasma colonies were demonstrated by Giemsa stain in the examined tissues. These results give a spotlight on the significance of *M. arginini* in she-camel.

Key words: Mycoplasma · Histopathology · She camel · Pneumonia · Reproductive disorders · Mastitis

INTRODUCTION

One humped camel (*camelus dromedarius*) is a multipurpose animal, which has enabled the nomadic people of the arid and semiarid areas of Africa and Asia to live in a difficult environment [1]. It can survive and flourish under tough and arid circumstances in the desert and nomadic areas, whereas it can survive for up to 20 days without any water, while cattle can survive without water for only 4 days. Camel is known also as a desert ship, with highly beneficial purposes, since; it produces milk, meat, hair and hides. Camels are less susceptible than cattle to endemic diseases and tolerant to heat and sparse rough pastures of the desert. In natural desert habitat, whereas camels are usually raised particularly during the long dry season, camels are subjected to severe stress conditions which render them susceptible to many diseases [2]. Although camels were considered in the past and for a fairly long time, as resistant to many disease causing factors [3], it has been proved that camels are susceptible, the same as other livestock or even more, to the common disease causing pathogens affecting other animal species [4, 5]. In Egypt, there has been increased interest in camel as an important source of milk and meat [6, 7].

Mycoplasma is a bacterium from the family Mycoplasmataceae lacks a cell wall and resistant to some antimicrobials, such as penicillin and cephalosporin, which act by inhibiting cell wall synthesis. Mycoplasma is smallest fastidious bacteria which can cause diseases in major species of animals including humans [8]. Transmission is usually by direct contact with respiratory secretions as well as aerosolization [9]. Mycoplasma causes major economic losses mainly by pneumonia, arthritis and loss of weight gain in calves, mastitis in cows and reproductive problems of both cows and bulls [10].

Corresponding Author: Abeer E. El-Metwally, Pathology Department, Animal Reproduction Research Institute (A.R.R.I.), Giza, Egypt. E-mail: drabeerwama@yahoo.com.

Little is known about the role of mycoplasma in the etiology of diseases in camels. This is partially due to the lack of investigations on the occurrence of mollicutes such as, Mycoplasma, Ureaplasma, or Acholeplasma, in camels [11]. Moreover, little data are available on the mycoplasma flora of clinically healthy camels [12].

Investigation on camel mycoplasmosis in Egypt was previously by some authors such as Ahmed [13], Sabry *et al.* [14], Fayad and Sabry [15], Sabry and Ahmed [16] and El-Shabiny *et al.* [17] who could isolate *M. arginini, A. laidlawi, Ureaplasma species* and untypable Mycoplasma species.

The present survey aimed to throw the light on the natural occurrence of mycoplasma and ureaplasma in she-camels from slaughter-house. The microbiological and histopathological studies of lung, uterus, cervix, vagina and mammary gland were to be investigated.

MATERIALS and METHODS

Collection of Samples: Tissue specimens were collected from 100 adult she-camels, slaughtered at Kerdasa Abattoir at 6th October governorate, Egypt. From each animal, 5 tissue specimens were taken (uterus, cervix, vagina and mammary gland parenchyma as well as lung). Animals were selected randomly among the slaughtered she-camels. The samples were examined visually for gross examination. Each tissue sample was divided into two parts, one part was put in a sterile polyethylene bag in an ice box under aseptic conditions for mycoplasma and ureaplasma isolation and the second part was immersed in 10% neutral buffered formalin saline for histopathological evaluation. The samples were collected through out 14-months from January 2009 to March 2010.

Mycoplasma and Ureaplasma Isolation and Identification: For mycoplasma isolation the exterior of the organ was sterilized by searing with a hot spatula. Small pieces of tissue were taken aseptically and best chopped with sterile scissor then placed in a separate sterile screw-capped bottle containing either mycoplasma or ureaplasma media. Isolation and identification of isolates were done according to Ruhnke and Rosendal [18].

Tissue Preparation for Histopathological Studies: The fixed specimens were trimmed, washed and dehydrated in ascending grades of alcohol, cleared in xylene, embedded in paraffin and sectioned at 4-6 u thickness, then stained with Haematoxylin and Eosin (HandE) stain and, when necessary, with special stain (Geimsa stain). The slides were examined using light microscope [19].

RESULTS

Mycoplasma and Ureaplasma Isolation: The isolation rate of *Mycoplasma arginini* from lung and vagina was 6 and 2% respectively. No mycoplasma was isolated from cervix, uterus and mammary glands. Also no ureaplasma was detected in all samples.

Histopathological Examination

Lung: Macroscopic lesions were recorded in 4 out of 100 lungs. Three lungs showed marbling appearance with hepatization and presence of abscesses containing cheesy material, while in the other three lungs revealed marked hepatization with sever congestion and their cut section oozes brownish exudates.

Microscopic findings of the examined lung tissues revealed several types of pneumonia. Three lungs specimen out of 100 animals showed diffuse suppurative bronchopneumonia in which bronchiolar epithelium showed necrosis with losing of cilia, desquamation within the lumen with peribronchial edema and lymphocytic infiltrations. Alveoli showed diffuse suppurative pneumonia which characterized by diffuse neutrophillic accumulations with severe perialveolar congestion (Figs. 1 and 2). Other alveoli revealed prominent emphysema. Interstitial tissue was thickened with edema and neutrophil and lymphocytic infiltrations accompanied by vascular congestion (Fig. 3). Pleura showed edema with perivascular lymphocytic infiltrations (Fig. 4).

Three lungs specimen out of 100 animals showed diffuse fibrinosuppurative pleuropneumonia with caseous necrosis in which necrosis, loss of cilia and desquamation of bronchiolar epithelium were observed while other parts showed hyperplasia. Perbronchial lymphocytic infiltrations and thickening of interstitial tissue, edema and mild hemorrhages accompanied by severe vascular congestion were observed (Fig. 5). Neutrophiles, macrophages and lymphocytic infiltrations were observed among interstitial tissue. Alveoli showed diffuse caseous necrosis with diffuse fibrinosuppurative pneumonia and fibrin thrombi within the blood vessel (Fig. 6). Caseionecrotic pleurisy was noticed with diffuse fibrin formation associated with mononuclear cellular infiltrations and neutrophiles as well as fibrin thrombi (Fig. 7).

Table 1: Type, numbers and percentages of pathological findings in different tissues from the examined she-camels:

Type of tissues	Type of lesion	No.	%
1-Lung	Diffuse suppurative bronchopneumonia.	3/100	3%
	Diffuse fibrinosuppurative pleuropneumonia with caseous necrosis.	3/100	3%
2-Uterus	Chronic granulomatus endometritis.	4/100	4%
3- Cervix	Diffuse and focal mononuclear infiltrations.	4/100	4%
4- Vagina	Granular vaginitis.	4/100	4%
5- Mammary gland	Acute diffuse purulent mastitis.	2/100	2%
	Chronic mastitis.	4/100	4%

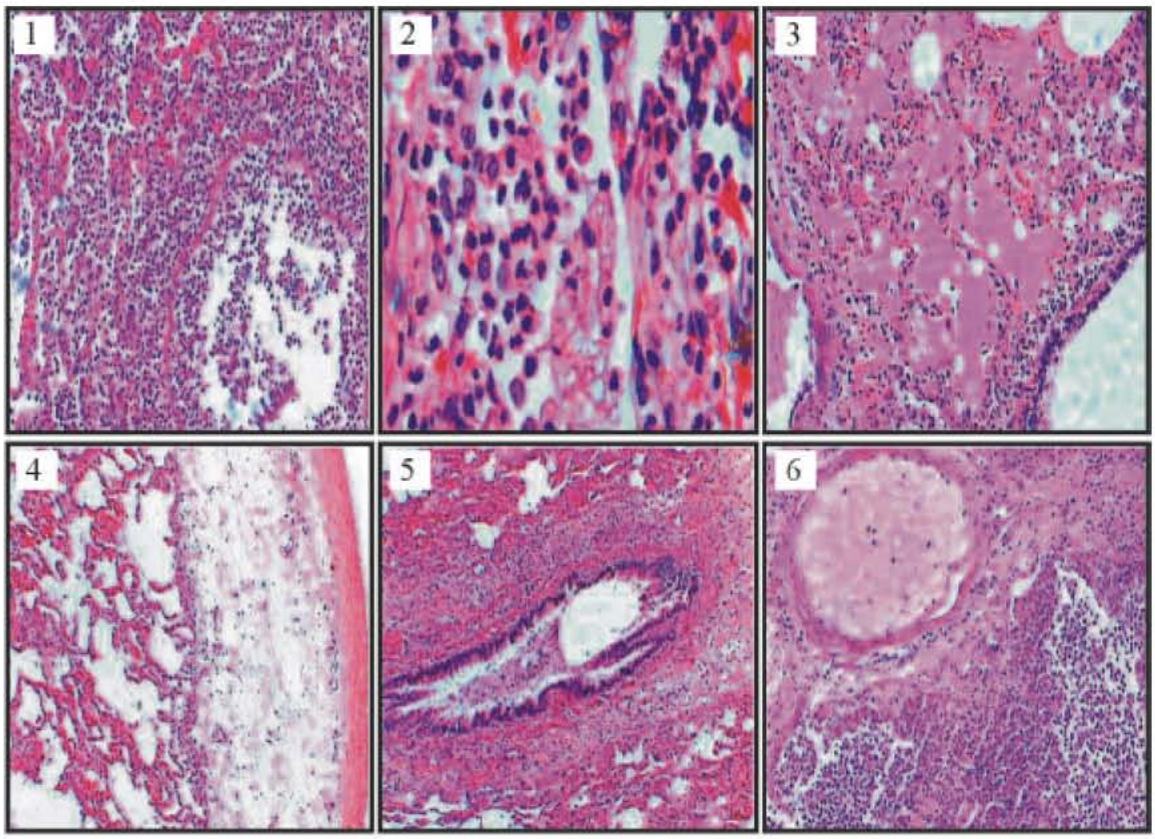

Fig. 1: Lung tissue showing diffuse suppurative pneumonia which characterized by diffuse neutrophilic accumulations within the alveolar lumen and perialveolar congestion (HandE Stain, X10).

Fig. 2: High power of the previous picture showing diffuse neutrophilic accumulations within the alveolar lumen and perialveolar congestion (HandE Stain, X40).

Fig. 3: Lung tissue showing thickening of interstitial tissue with exudate, neutrophil and lymphocytic infiltrations accompanied by vascular congestion (HandE Stain, X10).

Fig. 4: Lung tissue showing diffuse suppurative pleuropneumonia characterized by thickened pleura with perivascular lymphocytic infiltrations and edema. Parenchyma characterized by. perialveolar congestion, alveolar emphysema, thickened interstitial tissue with neutrophil and lymphocytic infiltrations (HandE Stain, X4).

Fig. 5: Lung tissue showing diffuse fibrinosuppurative bronchopneumonia characterized by necrosis, desquamation of bronchiolar epithelium, perbronchial lymphocytic infiltrations. Parenchyma characterized by perialveolar congestion and thickened interstitial tissue (HandE Stain, X4).

Fig. 6: Lung tissue showing diffuse fibrinosuppurative pleuropneumonia characterized by neutrophiles, macrophages and lymphocytic infiltrations among interstitial tissue and within alveoli with caseous necrosis and fibrin thrombi within blood vessel (HandE Stain, X10).

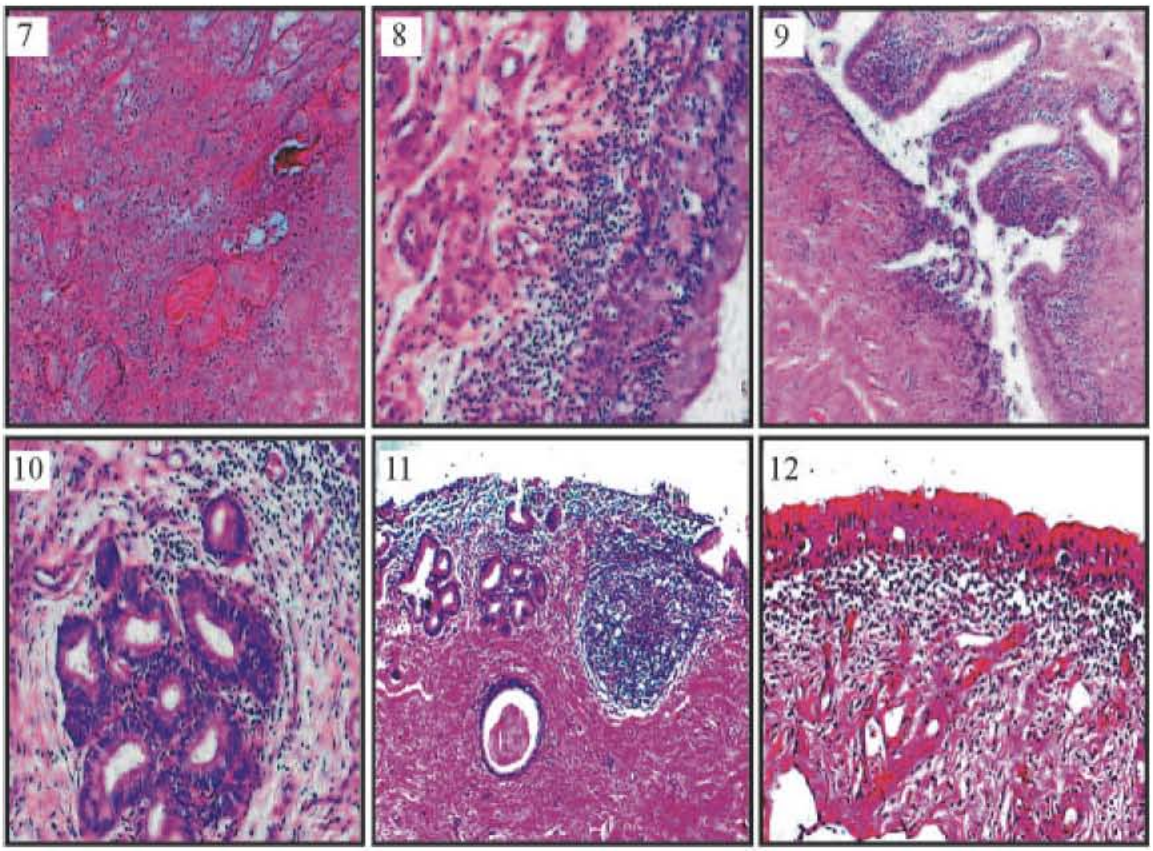

Fig. 7: Pleura showing caseionecrotic pleurisy with diffuse fibrin deposition accompanied by neutrophiles, macrophages and lymphocytic infiltrations as well as multiple fibrin thrombi (HandE Stain, X4).

Fig. 8: Uterine tissue showing intracellular infiltrations of the lining epithelium with lymphocytes and mast cells (exocytosis) and vacculation with subepithelial lymphocytic infiltrations (HandE Stain, X10).

Fig. 9: Uterine tissue showing neutrophiles, macrophages and lymphocytic infiltrations in the subepithelium with multinucleated giant cells and necrosis in some parts with periglandular lymphocytosis (HandE Stain, X4).

Fig. 10: Uterine tissue showing cyctic dilatation of uterine glands accosciated with periglandular cellular infiltrations (HandE Stain, X10).

Fig. 11: Uterine tissue showing desquamation of epithelial lining, subepithelial granulome formation and lymphocytic inflammatory cells infiltrations with fibrin thrombosis in blood vessels and glandular cystic dilatation (HandE Stain, X4).

Fig. 12: Cervical tissue showing exocytosis with lymphocytes and mast cells associated with vaculations and subepithelial lymphocytic inflammatory cells infiltrations (HandE Stain, X10).

Uterus: Gross appearance of uterine tissue of 4 animals showed congestion of mucous membrane with profuse purulent exudates within their lumens.

Microscopic examination of she-camel uterus revealed chronic endometritis. The Lining epithelium showed intracellular infiltrations with lymphocytes and mast cells (exocytosis) and vacculation (Fig. 8). The subepithelial layer was characterized by cellular infiltrations predominantly lymphocytes with few macrophages, neutrophiles, plasma cells, oesinophiles and multinucleated giant cells (Fig. 9). Degenerated endometritis with fibrosis resulted in atrophy of endometrial glands and the formation of nests associated with cystic dilatation were observed. Goblet cell hyperplasia of the glandular epithelial cells was noticed and other glands showed necrosis with periglandular lymphocytosis (Fig. 10).

Granulomatus endometritis were noticed within the uterine tissues in some cases which characterized by granulomatus focci, consisted of lymphocytes,

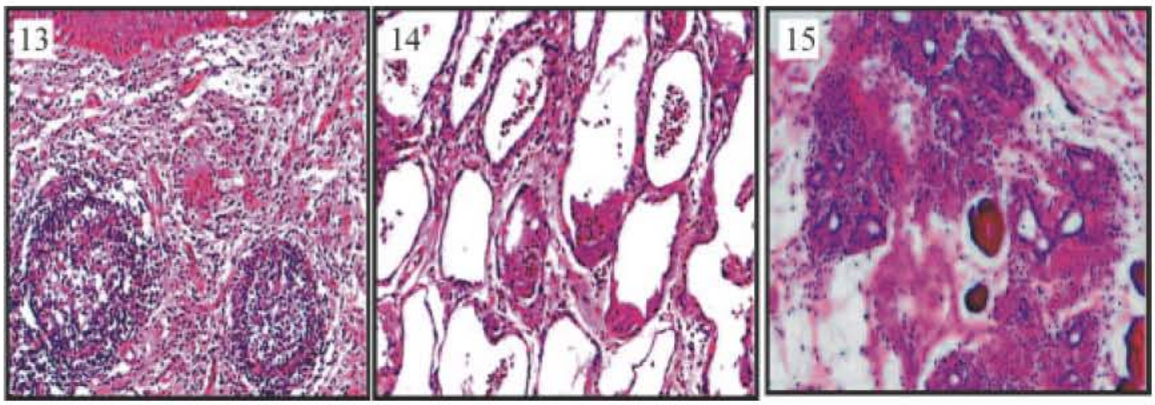

Fig. 13: Cervical tissue showing diffuse and focal lymphocytic and plasma cells infiltrations at the subepithelium (HandE Stain, X10).

Fig. 14: Mammary gland tissue showing acute diffuse purulent mastitis associated with infiltration of neutrophils and mononuclear cells in the lumen of acini and within the interlobular tissue (HandE Stain, X10).

Fig. 15: Mammary gland tissue showing chronic mastitis characterized by focal and diffuse aggregation of inflammatory cells mainly lymphocytes and macrophages, extensive alveolar atrophy and corpora amylacea (HandE Stain, X4).

macrophages, histiocytes and reticular cells with multinucleated giant cells situated mostly in the subepithelial layer. Fibrin thrombi in the subepithelial blood vessels was observed (Fig. 11).

Cervix: Macroscopically, the cervix of 4 out of 100 animals showed granular petechial hemorrhages with slight congestion and brownish mucoid exudates.

Microscopic examination of the cervical tissues revealed chronic granular cervacitis. The cervical epithelium showed exocytosis with lymphocytes and mast cells associated with vacuolations (Fig. 12). Diffuse and focal lymphocytic and plasma cells infiltrations at the subepithelium region and in the muscle layer were noticed (Fig. 13). Perivascular cuffing and vacuolar degeneration of endothelium were observed (Vasculitis).

Vagina: Grossly, the vaginal mucosa of 4 animals was congested, thickened due to scattered fine nodules rising on the mucosa forming granular vaginitis.

Microscopically, the vaginal mucosa showed diffuse lymphocytic infiltrations and lymphoid nodules in the subepithelial region of the mucosa as well as in the tunica muscularis. At places, the epithelium over the lymphoid nodules was exfoliated showed focal areas of hydropic degeneration with patchy desquamation of vaginal epithelium. Perivascular plasma cells and lymphocytic cuffing with vascular congestion were observed in the sub-mucosa as well as in the tunica serosa.

Mammary Gland: Mammary glands of 2 out of 100 animals were slightly enlarged and of moderate grey-yellowish colour. The other four examined glands appeared atrophied and firm in consistency with grey-whitish discoloration.

Microscopically, there were acute diffuse purulent mastitis associated with galactopheritis in two cases which characterized by infiltration of neutrophils and mononuclear cells in the lumen of acini and within the interlobular septa. Also, there was desquamation and degeneration of the epithelial cells of mammary acini. Pronounced homogenous eosinophilic contents and cellular debris were observed within the ducts (Fig. 14). Also, there were four cases of chronic mastitic which were characterized by chronic galactopheritis with extensive alveolar atrophy. The general patterns were alveolar epithelial necrosis with desquamation and destruction of epithelium in addition to marked vacuolar degeneration and cystic dilatation of acini. The glandular acini or lobules were replaced completely by fibrous connective tissue. Focal and diffuse aggregation of inflammatory cells mainly lymphocytes and macrophages with plasma cells and histiocytes were evident. Among this chronic fibrosing changes, corpora amylacea was a constant finding inside the lumen of mammary acini. Interlobular and intralobular ducts were mostly dilated and showed desquamation or hyperplasia of the lining epithelium with accumulation of macrophages, lymphocytes and plasma cells around the ducts. Squamous metaplasia of epithelium of the ducts was common finding (Fig. 15).

Mycoplasma organisms were demonstrated by Giemsa stain in all examined tissues.

DISCUSSION

Mycoplasma is a group of microorganisms belonging to class Mollicutes, which characterized by lack of a rigid cell wall [20]. Mycoplasma has variable surface lipoproteins allowing for bacterial adhesion and evasion of the immune system. Moreover, it can induce apoptosis of lymphocytes and hinders neutrophil activation [8].

In this work, the collected tissue samples were stained by Giemsa stain to confirm the laboratory bacteriological results. We noticed that the percentage of pathological lesions due to *Mycoplasma arginini* was isolated from lung and mammary gland of slaughtered she-camels was 6%. Meanwhile it was 4% in samples from uterus, cervix and vagina. Such lower percentage of isolation than that from mycoplasma isolation might be attributed to the fact that isolation from chronically affected animals is sometimes difficult, because of the overgrowing of bacteria of secondary infections or the inhibitory effect of the administered antibiotics [10].

Mycoplasma can colonize on respiratory epithelium and attach to the cilia if they are not cleared away by normal defense mechanisms such as airflow, competition by normal resident flora, mucociliary clearance or phagocytosis. It has many mechanisms for attachment including fimbriae, molecular attachments (hemagglutinins) and adhesions (M proteins) or by physical means (electrostatic charge) [21].

Caswell and Williams [22] reported that the most susceptible site for mycoplasma colonization is the junction of the alveolar ducts and the gas exchange region as there are fewer mucous cells and cilia.

In this study, results of gross changes and microscopic examination of lung were in agreement with those described by Elfaki *et al.* [12], Rodriguez *et al.* [23], Egwu and Aliyu [24], Ajuwape *et al.* [25], Gagea *et al.* [26], Fatma *et al.* [27] and Mohamed and Abd-EL Salam [28]. Pathogenesis of necrotic-suppurative foci in lung was explained by Hum *et al.* [29], Edy and Joachim [30] and Steven *et al.* [31] as massive presence of mycoplasma resulting in production of necrotizing factors followed by severe oxidative damage of tissues due to its ability to generate a significant amount of hydrogen peroxide which has an important role in pathogenesis and its virulence. Macgavin *et al.* [32] mentioned that the interstitial bronchopneumonia as remarkable findings in mycoplasma infection caused by immune suppression, cilia dysfunction and unregulated production of tumor necrosis factor-alpha (TNFα).

There was either fibrinous or fibrinocellular within the alveolar lumina. These findings were concomitant with those given by Fatma *et al.* [27] and Macgavin and Zachary [33]. In this respect, the production of fibrin explained by Jubb *et al.* [34] due to development of inflammation and the increase of blood vessels congestion so widening of blood vessel wall pores which allowed fibrin to escape from blood to pulmonary tissue under the effect of bacterial toxins. In addition to the increased permeability of the blood vessels, increased procoagulant activity and diminished profibrinolytic activity of the lung, more fibrin is present which is chemotactic to leukocytes [27].

Gagea *et al.* [26] and Shahriar *et al.* [35] explained that the pulmonary necrosis incited by mycoplasma colonization occurred as a result of vaculitis and thrombosis which initiated by the damage effect of the toxins on endothelial lining of blood vessels.

Mycoplasma has been considered as a cause of many types of genital infectious diseases [36]. The granular vaginal mucosa noticed in this work was specific to mycoplasmosis. The gross and microscopic lesions in the vagina, cervix and uterine tissues were almost similar, but vary in their severity to those observed by other workers [37-40] in natural and experimentally mycoplasma infections. Rana *et al.* [39] and Rodriguez *et al.* [41] added that the genital tract is one of target organs of mycoplasma which attaches to the genital epithelium and causes damage through its toxins. Moreover, Crouse *et al.* [42] speculated that mycoplasma could increase inflammation by stimulating macrophages to produce TNFα and inducible nitric oxide synthesis because of their ability to interact with the host immune system. The close contact between mycoplasma and the target cell membrane resulting in metabolic components exchange from mycoplasma to host cell which enable oxidative damage of host cells [43]. In this study, presence of vascular and endothelial damage as well as thrombosis followed by degenerative changes was observed. These changes were explained by Rodriguez *et al.* [36] to the presence of periglandular and perivascular fibrosis under the effect of mycoplasma toxins. The presence of large number of lymphocyte and plasma cell infilterations in the epithelial, sub-epithelial, muscular and serosal layers and around the blood vessels in the genital tract of the infected animal indicates that the strong cell-mediated responses are directed against the invading organism [44].

Chronic endometritis, cervicitis and vaginitis observed in the present study were explained by Trichard *et al.* [40] and Lu *et al.* [45] as a result of vasculitis which interfering with the blood supply

ending with chronic necrotizing inflammation. However, the vacuolar degeneration of the epithelial cells lining the mucosa of vagina indicates that myoplasma causes damage to the epithelial cells. Mycoplasma have a special affinity for secretory epithelial surfaces, whereas, they get intimately attached to sialic acid receptors present on host cells, causing damage to host cells by various mechanisms [44].

Gross and microscopic mammary gland findings were coordinate with Kumar *et al.* [38], Darzi *et al.* [46], Rodriguez *et al.* [47] and Byrne *et al.* [48]. These findings explained by Darzi *et al.* [46] and Razin [49] as the epithelial cells of acini and lactiferous ducts are the target for mycoplasma whereas it attaches to the acinar epithelium. Then damage occurs by its toxins and metabolites as H_20_2 and hydrolytic enzymes such as protease and phospholipase. In addition, mycoplasma utilizes the fatty acids and cholesterol of cell membrane. Diffuse infiltration of neutrophils and mononuclear cells within the lumen of acini and in the interlobular septa in addition to degeneration and desquamation of alveolar epithelium were observed in this study. In this respect, Byrne *et al.* [48] and Paape *et al.* [50] mentioned that neutrophils play a vital role in initiating the early inflammation in mycoplasma mastitis through phagocytosis and destruction of the invading pathogens by releasing of chemicals. The latter induce swelling and sloughing of secretory epithelium as well as decreased their secretory activity. Resident and newly migrated macrophages help to reduce the damage of the epithelium by phagocytosing neutrophils that undergo programmed cell death through a process called (apoptosis).

In this work, chronic mastitis was manifested by increased fibrosis with the resultant atrophy of the glandular parenchyma accompanied by increasing of the interlobular collagen fibers that replaced the glandular tissue. These results are consistence with those obtained by Jensen and Swifts [51], Baluk *et al.* [52] and Braz [53]. The squamous metaplasia of ductal epithelium observed in this study was in accordance with that of Banga andGupta [54] who decided that it caused by the chronic irritation of the persisting exudates in the ductal lumina.

In conclusion, results of the present study stressed on the importance of *Mycoplasma arginini* in she-camel pneumonia, reproductive disorders and mastitis.

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