Global Veterinaria 7 (2): 129-137, 2011
ISSN 1992-6197

Histopathological and Microbiological Studies on the Teat Affections in She-Camel

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Abstract: fifty adult she camel were examined for teat abnormalities at kerdasa slaughterhouse at 6th October governorate, Egypt. Teats were examined pathologically and microbiologically. Gross examination of teats revealed 76.5% showed teat lesions. The total bacterial recovery rate was 36% and the isolated bacteria were S. *aureus* 14.5%, *Sc. spp*, 18%, *E. coli* 14%, *CNS*, 4%, *Bacillus species* 1.5%, *P. hemolytia,* 1.5% and *P. aeruginosa* 1.5 %. While total mycotic recovery rate was 26.5% with isolation of *C.albicance* 9.5%, *A.Fumigatus* 9%, *A.niger* 2%, *mucor* 2.5% and *cladosporium species* 8.5%. Histopathological examination of teats revealed two types of thilitis, Necrotic suppurative theilitis (25.5%) and Necrotic non suppurative theilitis with diffuse fibrosis (38%).From our results we concluded that: *S. aureus* and *C.albicance* are the most prevalent isolates of she camel theilitis. A positive correlation in this work was suggested between tick infestation and teat end callosity which are predisposing to theilitis.

Key words: Histopathology · Bacterial Theilitis · Mycotic Theilitis · She- Camel

INTRODUCTION

Camels are important source for milk production in desert societies, their milk supposed to have medicinal properties, as it containing insulin like protein so it has hypoglycemic effect. In Egypt the length of lactation in she camel varies from 8 to 18 months, with mean daily yields ranging from 10- 22 kg, this range could be reduced with mastitis [1-3]. Camel's milk usually consumes in the raw state without heat treatment, so it has public health hazard on consumers with infected teat. Teat skin, teat orifice and their environment are the main reservoir for entrance of opportunistic pathogens into the mammary gland. Also they act as a primary defense barrier against mastitis. There defense function is due to it lined with keratin that inhibits bacterial invasion and colonization. Keratin bacterial inhibition character is through opsonizing bacteria, neutralizing its toxins and preventing its adherence by its lacteal immunoglobulins. More over keratin contains fibrous proteins and fatty acid that electrostatically binding with pathogens cell wall to be more susceptible to osmotic pressure [4, 5].

Theilitis in dairy animal is referred to the inflammation in one or all of teat wall layers including teat skin, teat canal and cistern. Many different pathogens have isolated from camel's mastitic mammary glands, either bacteria or fungi in pure or mixed infection [6]. Pathogens can invade the teat canal ascending toward the mammary parenchyma, then colonize, multiply and produce there toxins and finally predisposing to mastitis. So teat skin should be free from microbial contamination/lesions to maintain the health of animals [7, 8]. There is a relationship between teat end callosity (teat skin lesions, hyperkeratosis) and theilitis, which could be caused by mechanical and circulatory impairment of the teat tissue, during milking process [9, 11].

This work was carried out for identification the histopathological changes of teat tissue in relation to isolated causative pathogens .

MATERIALS AND METHODS

Collection of Samples: A total of 200 quarter teat tissues of 50 adult slaughtered she-camel were collected from Kerdasa slaughterhouse at 6th October governorate, Egypt. Animals were selected randomly among the adult slaughtered she-camels. The samples were examined visually for gross examination for lesion and external

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parasites. 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 bacterial and mycotic isolation and the second part was immersed in 10% neutral buffered formalin saline for histopathological evaluation. The samples were collected through out 6-months from June 2010 to December 2010.

Microbiological Examination

Bacteriological Isolation: The collected specimens were cultured into nutrient broth for 24 h at 37°C. A loopfull was inoculated onto the following media, 5% defibrinated sheep blood agar, MacConkey and nutrient agar. The suspected colonies were picked up and subcultured onto the selective media for identification by microscopic examination and biochemical reactions using standard bacteriological methods according to Quinn *et al*. [12].

Mycological Examination: This was carried out according to Anaiscie *et al*. [13] Sabouraud's dextrose agar (Difco) plates containing 0.05 mg/ml Chloramphenicol (to inhibit bacterial growth) were prepared. The inoculated plates were incubated at 25°C for 7 days. Examination of the plate was done daily for any fungal growth. Representative colonies were subcultured on Sabouraud,s dextrose agar slopes with antibiotic for further identification.

Identification of Isolated Moulds: Identification of isolated moulds was based on their growth rate, temperature and colonial morphology. Then it was confirmed microscopically by the type of hyphae and fruiting heads according to Quinn *et al*. [12]. Identification of yeasts: Yeasts were identified to genera according to their microscopic morphology on corn meal or rice agar and to species according to their pattern of fermentation and assimilation of sugars. A pure yeast colony was streaked onto the surface of corn meal agar plates and the streaks were covered with sterile cover slips. After 2-3 days incubation at 37°C the plates were examined directly [12].

C-*Tissue Preparation For histopathological examination:* Specimens from teat of she camel were routinely processed, embedded in paraffin wax, sectioned at 4u and stained with Hematoxylin and Eosin and Periodic Acid Schiff stain (PAS) [14].

RESULTS AND DISCUSSION

Microbiological Results: In this study the total microbial recovery rate from she camel teat quarters revealed 62.5% adding to 14.5% of mixed infection of bacteria and fungi

Bacterial Examination: As shown in Table (1) the bacteriological examination of she camel teat quarters with pathological lesions in the present study revealed, isolation of S. *aureus* 29 (14.5%), *Streptococcus spp*, 36(18 %), including *Sc. agalactiae 28(14%), Sc.uberis 8(4%),E.coli28(14%), CNS 8(4%),Bacillus species3(1.5%),P.hemolytica 3(1.5%*) and *Ps. aeruginosa* 3 (1.5 %) The current study clarified that the total bacterial recovery rate was 36 % and the most prevalent isolated bacteria were *S. aureus, Sc agalactiae and E. coli* with isolation incidence of 14.5%, 14%, 14% respectively. These findings are nearly similar to that reported by Abeer El-Metwally, [15], Abdurahman *et al*. [16], from udder of slaughtered she camel and Kalla *et al*. [17], Kotb *et al*. [18],Saleh and Faye, [19]and Amena *et al*. [20] from milk of slaughtered she camel . While Fragkou,*et al* [21] was isolated the same organisms from the teat of ewes. This result illustrated by Mork *et al*. [22], Sharif *et al*. [23] who reported that those three organisms are the most common contagious environmental pathogens in dairy animals. They associated with unsanitary environment and the primary reservoirs of them are animal's environment, skin of udder and teat, in the presence of teat skin lesions, they ascend via teat canal. Abdel Gadir *et al*. [24], Piccinini *et al*. [25] added that *S. aureus* able to survive on teat skin and contaminates milk with an enterotoxin that pasteuralization resistant, so leading to food intoxication.

Table 1: Isolated bacteria from examined she camel teat quarters

	Bacterial species	No. Of samples	% from total (200)
A-Single infection:	Staphylococcus aureus (S.aureus)	21	10.5
	Streptococcus agalactiae (Sc. agalactiae)	17	8.5
	E.coli	15	7.5
B-Mixed infection:	S. aureus + Sc. uberis + Bacillus spp.	3	1.5
	S. aureus + Sc. agalactiae + E.coli	5	2.5
	P.hemolytica + CNS + Sc. agalactiae	3	1.5
	CNS + E.coli + Sc. Uberis	5	2.5
	E.coli + Sc. agalactiae + Ps.aeruginosa	3	1.5
Total		72	36

*Incidence from total No. of 200 teat quarters of examined 50 she camels.

Table 2: Isolated yeasts and moulds from examined she camel teat quarters

	Fungus	No.of samples	% from total (200)
A-Single infection:	Candida albicance (C.albicance)	14	7
	Asperigillus fumigates (A.Fumigatus)	11	5.5
	Cladosporium spp	10	5
	Mucor	2	1
B-Mixed infection	Cladosporium spp.+ Aspergillus niger (A. niger)	4	2
	Cladosporium spp. + A.Fumigatus	5	2.5
	C.albicance + mucor	4	2
	C.albicance+ A.Fumigatus	3	1.5
Total		53	26.5

*Incidence from total no. of 200 teat quarters of examined 50 she camels

Table 3: Teat quarters lesions distribution among the whole she camel

Teat lesions among the whole animal	No .of samples	% from total (200)
One affected teat	72	36
Two affected teats	50	25
Three affected teats	18	9
Four affected teats	12	6
Total	152	76

*Incidence from total no. of 200 teat quarters of examined 50 she camels

Table 4: Types of skin and teat lesions in she camel :

Gross pathology	No .of samples	% from total(200)
Tick infestation and hyperkeratosis	116	58
Blind teats	3	1.5
Obstructed teat canal	5	2.5
Fistula and pus	11	5.5
Nodular formation	17	8.5
Total skin or teat lesions	152	76

*Incidence from total no. of 200 teat quarters of she camels teat lesions.

Mycological Examination: In this work as shown in Table (2) the total mycotic recovery rate from she camel teat tissue with pathological lesions represented 53(26.5%) out of 200 teat quarters of she camels. The mycotic isolates were *C.albicance* 21 (10.5 %), *A.Fumigatus* 18 (9%), *A.niger* 4(2%), *mucor* 6 (3%) and *cladosporium 19* (9.5%) as single or mixed infection. This result come in agreement with that observed by Abeer El-Metwally, [15],Kotb *et al.*[18] who isolated *C.albicance,A.Fumigatus and A.niger,* from udder and milk of she camel. On the other hand, Ahmed and Sotohy [26],Abeer and Hanaa [27]and Fadlelmula *et al.* [28] isolated the same fungi from udder, teat surfaces, milk of cows and buffaloes.

The present study showed that the most prevalent isolated fungi were *C.albicance* which were recovered from 11.5% of she camel teat tissues this result is nearly similar to that observed with Kalla *et al.*[17] and Hanaa *et al*.[29] from milk samples and udder of slaughtered she camel. The predominance of *C. albicans* was explained as *C.albicance* is normal commensals of human oral mucosa and normal inhabitants of the teat skin, when the local immunity of dairy animal is broken down they become virulent pathogens and causing outbreaks of mycotic mastitis. It also associated with environmental contamination and with public health significance, as its spores are pasteurization resistant [30- 32].

Histopathological Results

Gross Pathology (Teat End Callosity): As shown in Table (3) The incidence of teat gross lesions in this investigation among the whole animal was at, one teat (36%), two teats (25%), three teats (9%),and four teats (6%) and these results were coincide with Sharma *et al*. [7]. The higher prevalence in this work was noticed in hind teat quarter (54%) than the fore teat quarters (22%) and these observed results were agreement with that observed by Saleh and Faye [19].

As shown in table (4) out of 200 she camel quarter teats examined in this investigation 76 % teat skin lesions were detected on naked eye. This recorded rate is higher than that reported by Abera *et al.* [33] who detected 5.5% teat lesions on teats of she camel. Most of teat skin (58 %) were harbored several hard ticks of dark brown

Table 5: Histopathologicallesions of examined she camel teat quarters in relation to isolates

Histopathological Lesion	Isolated organisms	No. of samples	% from total (200)
Necrotic suppurative theilitis	S. aurous10, Sc. Agalactiae 9,Ecoli 7, Ps. aeruginosa 3,CNS 2, Sc .uberis8, P.hemolytica1	229	14.5
	adding to only mycoyic isolates C.albicance 9, A.Fumigatus 7, A.niger 4,mucor	22	11
Necrotic non suppurative theilitis with diffuse fibrosis	Sc. Agalactiae19, Ecoli 21, S. aureus 19, P.hemolytica2, CNS 6	43	21.5
	adding to only mycoyic isolates C.albicance 10, A.Fumigatus 11, mucor 3,Cladosporium spp 17	31	16.5

*Incidence from total no. of 200 teat quarters of examined 50 she camels.

Adding to14.5% is mixed infection of bacteria and fungi with mixed lesions

color intensively adherent to the teat skin. Teats infested by ticks were characterized by irregular, rough, scaly hyperkeratosed skin surface and dark pigmentation accompanied with skin lesions varied from erosions, ulcers to fistula formation with or without abscess formation. This result comes in agreement with that given by Abeer El Metwally [15], Biffa *et al.* [34] and Abdurahman *et al.* [16] they suggested appositive correlation between tick infestation and teat lesions due to unhygienic milking management. Also, teat skin is a predilection site for tick infestation which causes skin and teat lesions. The potential dangers of ticks are they facilitate bacterial entry and leaves behind permanent tissue damage to the udder leading to theilitis . Blind teats in this work were detected in three animals (1.5%) which less than the percentage reported by Abdurahman *et al*. [16] and Abera *et al*. [33] they reported 3.3%,33.8%, blind teats respectively,in she camels. Cut section inside teats revealed (21%) out of 152 were red congested mucosa (5.5%) of they showed fistula and pus. pale mucosa was detected in (31.5%) were (8.5%) of them showed nodular formation, these results were coincide with those observed by Mavrogianni *et al*. [35] in ewes teat.

Histopathological Lesions

Necrotic Suppurative Theilitis: As shown in table (5) this type was represented 14.5% (29 cases) Isolated bacteria from she camel quarter teats in this work were *S. aurous, Sc. agalactiae*, *Ecoli, Ps. aeruginosa, CNS, Sc. uberis, P.hemolytica* as single or mixed and in 11% (22cases) the most isolated fungi were *C.albicance*, *A.Fumigatus*, *A.niger,mucor* as single or mixed.

Microscopically: Characterized by fistula formation extended from epidermis to the dermis layer with subepithelial sinus filled with pus, in which the stratum cornium revealed extensive necrosis with mild hyperkeratosis. The germinal epithelium showed epithelial hyperplastic proliferation characterized by over thickening of the epidermal stratified epithelium in the form of dowengrowth folds or branching. The spinosa cell layers showed acanthosis, characterized by irregular over thinking. The prickle cell layer showed hydropic degeneration, necrosis with exocytosis. Tunica granulose was observed to contain coarse keratohyaline granules (Fig. 1). The subepithelial layer showed extensive suppuration and necrosis extended under the malpigian layer including the hair follicles and sebaceous glands. Also there were diffuse subepithelial cellular infiltrations mainly of dead PMN (polymorhoneclear cell), lymphocyte and macrophages accompanied with vasculitis and connective tissue necrosis were noticed in dermal layer (Fig. 2).

Lesions caused by fungi were positively stained yeast cell and fungal hyphae with PAS stain on fragmented keratine. The Sub dermal connective tissue showed necrosis with focal and diffuse inflammatory cell infiltrations mainly lymphocytes, plasma cells, macrophages, dead neutrophils, few eosinophils and scattered multineucleated giant cells (Fig. 3a,b). In addition to sever vasculitis (Fig. 4). The teat canal lining epithelium in most cases was showed, congestion, epithelial vacuolar degeneration and desquamations with subepithelial PMN and lymphocytic infiltration (Fig. 5). In other cases in which fungi were isolated teat canal lining epithelium rvealed hyperplastic and metaplastic changes into stratified squamos keratinized epithelium. Some parts of the canal showed ulcerations with subepithelial focal monocellular infiltrations. Diffuse necrosis of connective tissue surround the canal was also observed (Fig. 6) in which the fungal hyphae was positively stained with PAS within necrotic tissue,

Teat cistern showed vacuolar degeneration and desquamations of the lining epithelium with cellular infiltrations mainly PMN, lymphocytes and macrophages accompanied with necrosis, congestion and edema (Fig. 7) while in cases showed fungal isolation cisternal epithelium revealed degeneration and destruction in with

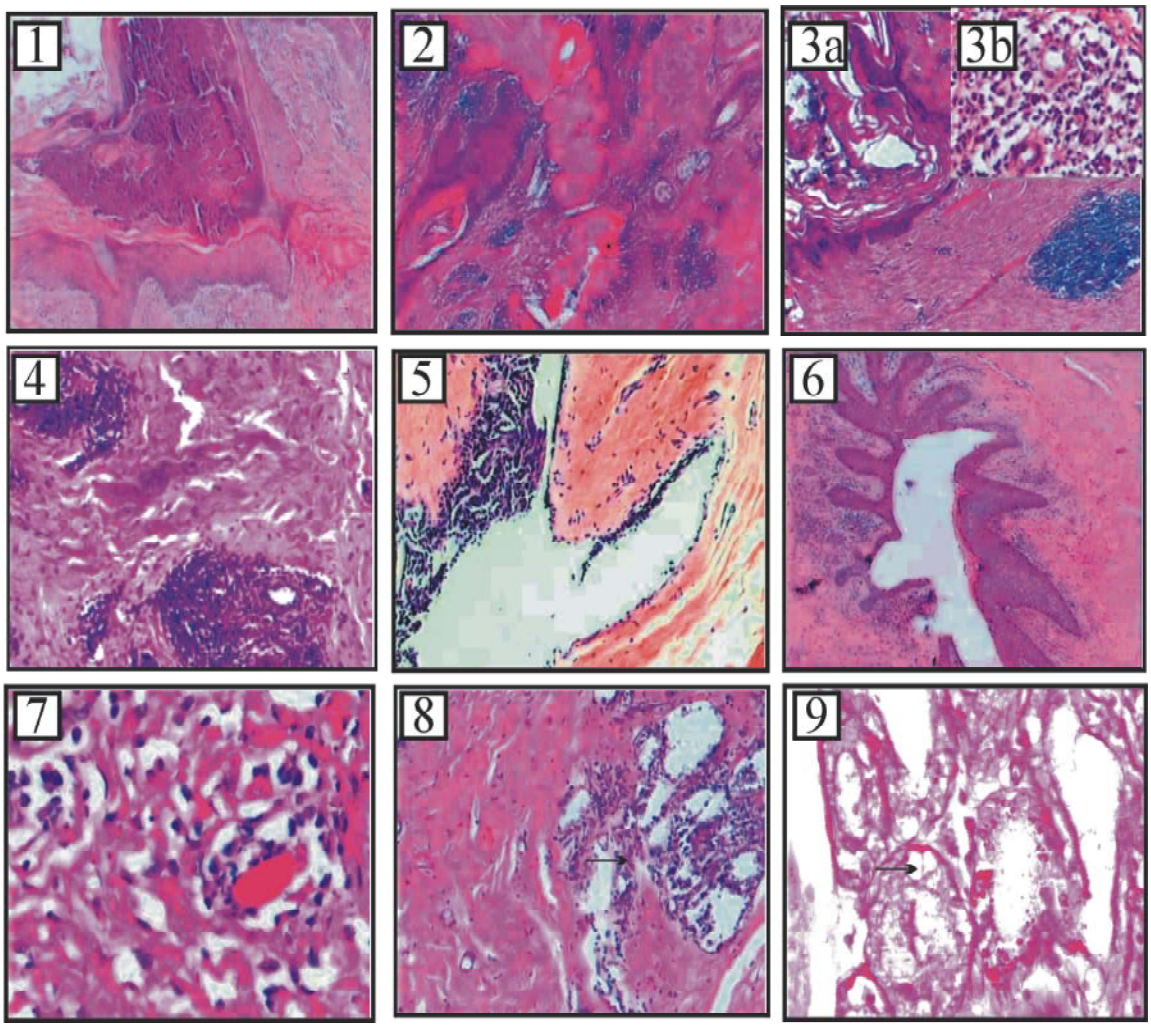

Fig. 1: She camel teat showingextensive necrosis and suppuration with fistula (HandE x4)

Fig. 2: She camel teat showing diffuse suppuration, necrosis and cellular infiltrations in the subepithelial layer (HandE x4)

Fig. 3a: She camel teat showing Prominent hyperkeratosise, acanthosis, extensive melanosis and focal mononeuclear cell infiltrations in subepithelial layer (HandE x4).

Fig. 3b She camel teat showing aggregation of neutrophiles,macrophages, lymphocytes, plasma cells and eosinophils together with multineucleated giant due to *C*.albicance (HandE x40).

Fig. 4: She camel teat showing sever vasculitis of the dermal connective tissue (HandE x10).

Fig. 5: She camel teat canal lining epithelium showing desquamations and subepithelial cellular aggregations (HandE x10)

Fig. 6: The teat canal lining epithelium showing hyperplasic, metaplastic changes, hyperkeratosis with acanthosis and subepithelial monocellular infiltrations, (HandE x10).

Fig.7 : Teat cistern lining epithelium showed vacuolar degeneration, desquamations with diffuse cellular infiltrations mainly neutrophils, mononeuclear cells (HandE x10)

Fig.8: Teat cistearne epithelial lining showed necrosis and desquamations with cellular infiltrations mainly lymphocyte and *C*.albicance appeared within its lumen arrow (HandE x10)

Fig.9: Teat cistearne showed *C*.albicance appeared within its lumen arrow (PAS stain x40).

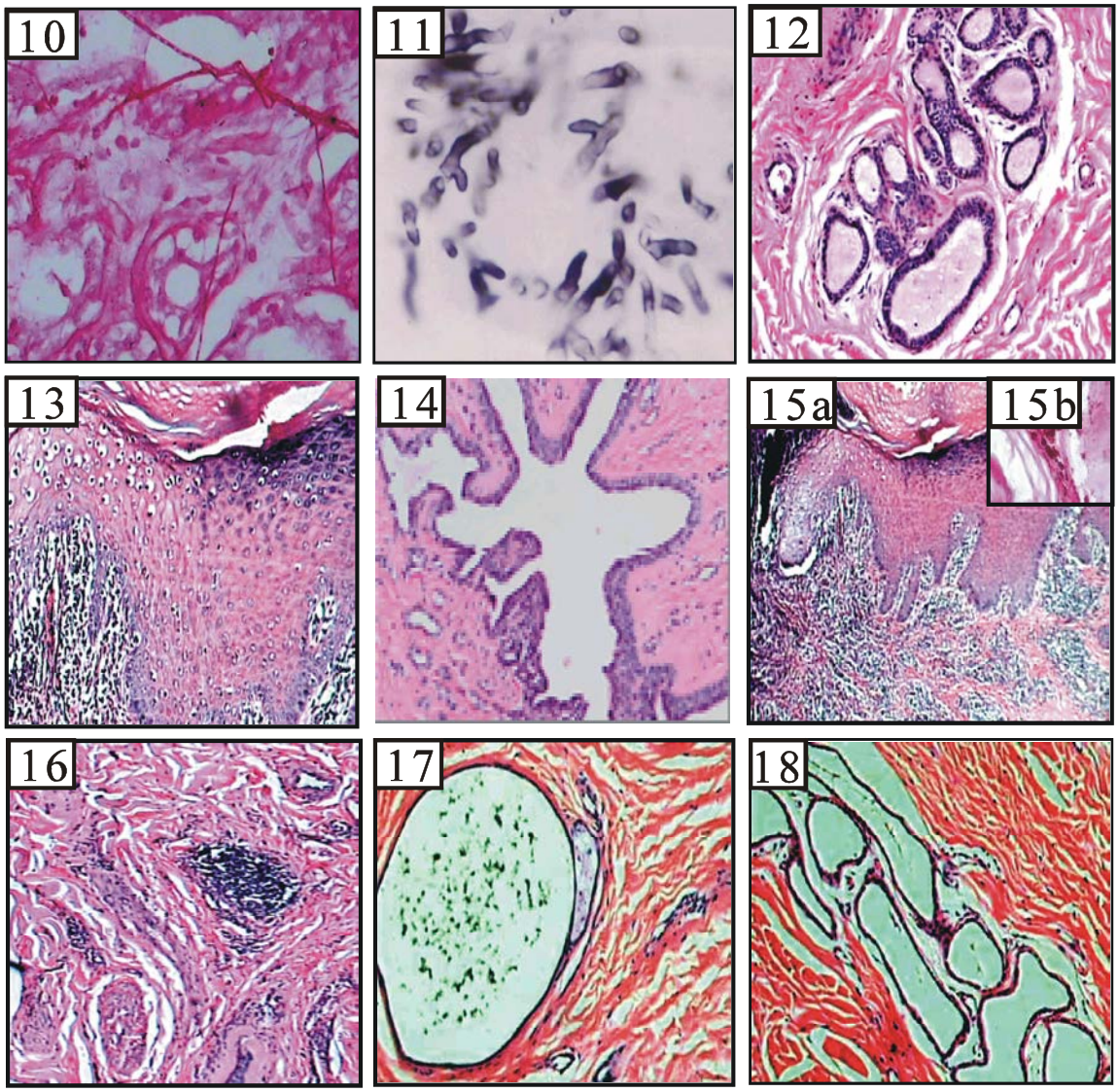

Fig. 10: Teat cistearne showed fungal hyphae appeared within its lumen arrow (PAS stain x40).
Fig. 11: She camel teat showing *A.Fumigatus* hyphae within necrosed subepithelial connective tissue (GMS stain x40).
Fig. 12: Rosette of Furstenberg in she camel teat showed diffuse cellular infiltrations mainly neutrophils, lymphocytes and macrophages with epithelial hyperplasia (HandE x10)
Fig. 13: She camel teat showing hydropic degeneration, vaculation and exocytosis in the prickle cell layer with subepithelial mononeuclear cell infiltration mainly lymphocytes (HandE x10)
Fig. 14: She camel teat showing, hyperplastic changes, acanthosis of teat canal lining epithelium (HandE x10)
Fig. 15a: She camel teat showing Prominent hyperkeratosise, acanthosis, hydropic degeneration, extensive melanosis with subepithelial monocellular infiltrations (HandE x4)
Fig. 15 b: *Cladosporium* spores were seen on the dead keratine with PAS stain x10
Fig. 16: The subepithelial connective tissue showed necrosis with focal mononeuclear inflammatory cells aggregation (HandE x10)
Fig. 17: She camel teat showed sebaceous glands cyst with diffuse fibrosis (HandE x10)
Fig.18: Rosette of Furstenberg showed cystic dilatation as island(HandE x10)

C.albicance present within destructed epithelium (Fig. 8). Fungal hyphae and *C.albicance* cell were detected positively stained with PAS and GMS stain within cistern,teat canal epithelium (Fig. 9,10,11).Those finding demonstrating that the main cause of thilitis could be mycotic only in those cases specially in absence of bacteria isolates from it . Rosette of Furstenberg revealed epithelial hyperplasia with cellular infiltrations mainly lymphocytes and macrophages (Fig. 12).

Necrotic non Suppurative Theilitis with Diffuse Fibrosis: This type of lesion represented 43case (21.5 %) of only bacteria were isolated *Sc. Agalactiae*, *Ecoli, S. aureus, P.hemolytica*, *CNS* .In addition to other 31 cases (16.5%) of only mycotic isolates which were *C.albicance*, *A.Fumigatus*, *mucor*,*Cladosporium spp* as single or mixed as showed in table (5).

Microscopically: Epidermis showed hyperkeratosis with over thickened fragmented keratin resulting in scaly appearance. Spinosa cell layer showed more pronounced acanthosis, hydropic degeneration, necrosis, extensive melanosis and exocytosis.

The germinal epithelium showed hyperplastic activity with increased mitotic figures (Fig. 13). The subdermal connective tissue showed diffuse fibrosis and mononeuclear cellular infiltrations resulting in sebaceous cyst formation. The teat canal lining epithelium was in most cases hyperplastic and metaplastic into stratified squamos epithelium accompanied with acanthosis in some parts (Fig. 14). In five cases (2.5%) there was unilateral obstruction of the dilated teat canal by thick keratin with excessive periductal fibrosis. Teat cistern and Fürstenberg's rosette showed mononeuclear cellular infiltrations mainly lymphocytes, macrophages and plasma cells. cisternal epithelium showed vacuolar degeneration, desquamations and pericisternal fibrosis as well as nodular proliferation and cystic dilatation in 2 of 5.

In cases infected with *Cladosprium spp* either mixed or single infection were characterized by the presence of brown *Cladosprium spp* spores . *Cladosporium spores* were seen on the dead keratine with PAS stain (Fig. 15a,b). The subepithelial Connective tissue characterized by fragmentation,necrosis with focal cellular infiltration formed mainly of lymphocytes, plasma cells,macrophages, with vasculitis and muscle necrosis (Fig. 16), Some hair follicles showed destruction. Fungal hyphae given positive with GMS stain within necrotic connective tissue. Also there were noticed fibrosis resulting in, sebaceous glands cyst (Fig. 17). Teat cistern and Rosette of Furstenberg showed cellular infiltrations, vacuolar degeneration, desquamation and diffuse fibrosis around it forming cystic dilatation island like. (Fig. 18).

Histopathological changes noticed in bacterial caused theilitis through the epithelial and sub epithelial malpigian layer of she camel quarter teat, were come in agreement with findings of Mouli, [36], Kiossis *et al.* [37], Abeer El Metwally [15], Mavrogianni *et al*. [35] and Kiossisa *et al*.[38]in teat of dairy cows, she camel and ewes,they attributed those changes as a response to toxic injuries due to mild reaction of tick spot which leads to continues irritation. This tick spot undergo secondary bacterial infection from the surrounding environment by pyogenic agents resulting in diffuse epidermal suppuration and necrosis extending to dermis. Hyperkeratosis which is noticed could be attributed to the chronic irritation from prolonged heavy tick's infestation of the teat together with defense trials from affected tissue. A degenerative change in the form of hydropic degeneration with necrosis were seen in the hyperplastic epithelium which suffered from toxic, hypoxic effect at the site of tick infestation. The hyperplastic activity and increased mitotic figures seen in the germinal epidermal layer that undergo dowengrowth folds into the underlying dermal connective tissue were referred as a regeneration process. While microscopic lesions in the teat canal, teat cistern and Rosette of Furstenberg were explained as due to traumatic lesion of the teat canal resulting in the formation of granulation tissue. Diffuse epidermal, dermal suppuration and necrosis noticed in this work are due to extracellular enzymes and cytotoxins that produce by secondary bacteria which aggravate the condition. Those cytotoxins are responsible for tissue adherence, invasion then necrosis of the teat epithelium and lactiferous sinuses. [30] and [40]. The unilateral keratin obstruction of the teat canal observed in this study were similar to Nickerson, [4] findings who explained it by keratin obstruction of the teat canal leads to dilatation of the ducts, retention of milk and secondary bacterial infection. So the keratine plug might be to prevent multiplication and upward movement of bacteria to mammary gland parenchyma

Histopathological lesions described in cases caused by fungi in the present investigation were coinciding with that observed by Jones and Hunt [40], jubb and Kennedy [41] and Abeer El Metwally [15]in teat of cattle and she camel who mentioned the dermal suppurative lesions and multinucleated giant cells as pathognomic lesion to mycotic theiltis. They also explained these histopathological lesions as a primary mycotic infection

penetrating through minute lesions evoked by the tick parasite irritation, that extend to the depth of hair follicles and sebaceous glands,resulting in epithelial necrosis. More over this tissues suppuration and ulcerations were attributed by El-Naggar *et al.* [42] and Kuhn and Ghannoum [43] to the toxic products of the fungi as acid proteinase of *C. albicans* and fungal proteolytic enzyme as well as mycotoxins (aflatoxin and gibberelin)which facilitate the entry of the organism from the colonization site into the parenchyma and cause necrosis . On the other hand giant cells play an important role in phagocytosis of the fungal elements.

Conclusions: From the present study we can be concluded that:

- *S. aureus, C.albicance* are the most prevalent of causes of theilitis she camel.
- Appositive correlation in this work were suggested between tick infestation, microbial isolation and teat end callosity which are predisposing to theilitis .
- Presence of giant cell, vasculitis and necrosis of teat indicates mycotic theilitis

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