

Ischemic Necrosis Of The Teat In Buffaloes With Its Effect On The Future Productivity

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ABSTRACT

Three hundred and forty buffaloes admitted to the department of surgery faculty of veterinary medicine Zagazig Univ. from October 2002- July 2007 with teat affections. The incidence of buffaloes affected with ischemic teat necrosis were 38.2%. It was commonly observed in autumn and winter, few weeks postpartum between the first and third lactation season. There were 255 teats with ischemic necrosis investigated where more than one teat may be affected in the same animal. The medial aspect of the base of teat especially of those hind teats was the primate site. The condition was examined and classified into six different forms according to the progress of the lesion. The initial changes were edema and thickening of the overlying skin which became necrotic and deep ulcerated. The necrosis extended to include the full-thickness of the teat and in some cases the whole teat. Different methods of treatment were used according to the stage of ischemic necrosis diagnosed. The results of treatment and fate of these different forms of the condition were recorded.

INTRODUCTION

In Egypt, buffaloes contribute the main dairy animals. Buffalo's milk is more valuable and farmers receive at least twice the price relative to cow's milk. Therefore, the integrity of teat and udder is important for that purpose. The anatomical position and physical effects of being milked and handled two times daily, the teats are subjected to a wide variety of disorders. Also some systemic infections and other injuries are accompanied with teat lesions (1). Different congenital and acquired teat affections of buffaloes were discussed (2-5). The apparent increase in the incidence of ischemic necrosis of teats in addition to its very stubborn treatment and bad results in shedding of the teat was the aim to study the details of this disorders; incidence of occurrence, causes, signs, diagnosis, prognosis and treatment.

MATERIAL AND METHODS

The buffaloes admitted to the department of surgery faculty of veterinary medicine from October 2002- July 2007 with teat affections were investigated. From which a focus was thrown on those affected with ischemic necrosis. The incidence of this affection in relation to the other teat affections, number of affected teats, site on teat and

degree of ischemic necrosis in addition to the seasonal of occurrence were recorded.

The history of this disorder was taken and the animals were clinically examined. The affected teats were subjected to: 1) visual inspection to describe color, shape, size and location of the disorder present; 2) careful palpation and rolling of the affected teat between thumb and finger to determine the degree of tissue destruction; 3) hand milking to observe milk flow and 4) probing the teat and gland sinus with teat siphon to detect if obstructing tissue in area.

Restraint and anesthesia were important for adequate examination and treatment. The animals were sedated by 0.05%mg/kg I.V. (Rompun 2% Bayer) and restrained in lateral recumbence. The four limbs were secured together by casting ropes where the udder and teats were fully observed behind. Local anesthesia was performed in cases of massive debridement and teat amputation by circumferential injection of 20ml of a lidocaine hydrochloride 2% at the base of the teat in the area of the annular ring.

Skin scraping from the periphery of ulcerated lesions was collected in clear dry Petri dishes containing 0.9% saline for the detection of microfilaria which stained by Giemsa's stain (6).

Treatment was performed according to the degree of necrosis and loss of tissues:

*In early diagnosed cases, where the teat was enlarged edematous with small blackish dry rough thickened areas of skin; cold and hot fomentation with povidone iodine 2% and Furaderm ointment (Amoun pharmaceutical Co. Egypt) were applied three times daily for five days. Systemic non-steroidal anti-inflammatory of Dicloflame 2.5% (Unipharm, El-naser Co.); 15 ml. I.M. for three days was used.

* In superficial and deep ischemic necrosis, where thin or thick brown scab cover unhealed ulcer; minimal debridement and rinsing by normal saline was used. Any necrotic, contaminated or infected tissue must be removed by curettage. Povidone iodine 10% swabbing and topical antibiotic spraying by Tetravet Aerosol, (Bonac Laboratories LTD, NewZeland) were used until complete healing.

*When ischemic necrosis extended to the full-thickness of teat observing teat sinus or udder cistern; massive debridement and rinsing were used. All necrosis was removed by scalpel until the healthy tissue was observed. The produced wound was partially closed or allowed to heal by second intension. In these cases purulent mastitis of the corresponding quarter was accompanied, systemic antibiotics in addition to drainage and daily dressing was important for treatment.

*When ischemic necrosis extended to the whole teat length except teat tip; the leathery thickened surrounding overlying necrosis was excised with care to the teat sinus. Teat fistula may be a sequel. Daily dressing and bandaging until healing was performed.

*When ischemic necrosis included the whole teat, amputation of that teat and drainage of the quarter infected were applied. After preparation of the surgical field aseptically and local anesthesia, the incision was made in the healthy tissue at the base of the teat. By removing the teat circumferentially, complete drainage of the infected milk was assured. Hemorrhage was controlled by ligation and

the teat lumen retained by continuous sutures in the wall (skin to mucosa) using interlocking mattress to maintain drainage.

Postoperative management included passive milk drainage every second day during the period of treatment. Intra-mammary infusion antibiotic, (Synulox, Pfizer Inc, NY, USA) every second day was administered when the interference reached the teat sinus. Systemic antibiotics, combination of penicillin & streptomycin (Pentomycin, Univet LTD, Ireland, 4ml/100kg I.M.) for five days was used in cases accompanied by systemic illness and mastitis or opening the teat sinus. Ivermectin 0.2mg/kg S.C. (Ivomec super, Merial, Netherlands) was injected when microfilaria was diagnosed. Sutures were removed ten days post-operative.

RESULTS

Three hundred and forty buffaloes admitted to the department of surgery faculty of veterinary medicine Zagazig Univ. from October 2002- July 2007 with teat affections. The incidence of buffaloes affected with ischemic teat necrosis were 38.2% (Table 1). It was commonly observed in autumn and winter (Fig 1), few weeks postpartum between the first and third lactation season (Fig 2). There were 255 teats with ischemic necrosis investigated where more than one teat affected in animals (Table 2). The disorder was observed commonly at the base of the teats especially those of the hind teats (Table 3). The ischemic necrotic teats were classified according to the wideness and deepness of tissue included in the teats (Table 4) into:

**Telitis with ischemic necrotic areas*

The teat appeared enlarged edematous with overlying blackish dry rough thickened areas of skin. The udder and rest of teat skin turned reddish-purple with some desquamation producing a dirty serous exudates (Fig 3). Deep fissures may be developed (Fig 4). This stage is the early form and often highly irritant to the animal. Local and systemic treatment of these cases had been proved effective without complication to the teats. The cases accompanied by mastitis was also cured.

***Superficial ischemic necrosis included the skin of the teat**

The lesion was commonly observed at the teat base and mostly on its medial side (Fig 5). The lesion was usually appeared as a small necrotic spot which extends further and circumscribes the root of the teat (Fig 6). Healing in these cases was completed by minimal unnoticed scar and the milk flow was normal.

***Deep ischemic necrosis included the skin and intermediate layer**

These cases were characterized by thick brown scab cover which on removing leaving unhealed ulcer at its center and blood oozes from the lesion. Some of these ulcers were accompanied by other ulcerative lesions in limbs (Fig 7). More than one teat was affected (Fig 8). Skin scraping revealed the presence of microfilaria in 16 case. The outcome of treatment resulted in a moderate scar at the area of lesion and the teat was obliquely elevated without impaired milk flow.

***Full-thickness teat ischemic necrosis**

The lesion extended to all layers of teat at its base observing the mammary tissue (Fig 9a) and teat sinus (Fig 10a). Mastitis in these cases was supervened. The wide surgical area was partially closed after removal of all necrosis (Fig 9b,c). The chronic old cases complicated by unhealthy granulation tissues in the site of lesion, were allowed to heal by second intension after daily dressing (Fig 10b).

Healing in these cases was completed by large scar deform the teat (Fig 10c). The corresponding quarter will eventually dry off as the result of secondary infection and mastitis.

***Ischemic necrosis included circumferentially the teat length except the teat tip**

The surrounding teat skin covering except the teat tip was leathery thickened. A line of demarcation with partial sloughing from the udder was noticed at the teat base. The end of the teat was healthy and function (Fig 11a). Removal of overlying surrounding necrosis resulted in loss of skin circumferentially from the teat (Fig 11b). The teat sinus was reached and therefore a fistula was a sequel in nine cases. Complete healing with contraction and epithelialization was accomplished within two months (Fig 11c). Impaired milk flow was noticed in six cases and slightly impaired milk flow was noticed in other cases.

***Ischemic necrosis of the whole teat**

The teat was voluminous leathery hard dark brown with line of demarcation from the udder (Fig 12a). Impaired milk flow was observed. Mastitis was accompanied in all cases. Amputation was the sole of treatment for teat and drainage of the infected quarter (Fig 12b). The corresponding quarter will eventually dry off as the result of secondary infection and mastitis.

Table 1. The incidence of teat affections in admitted buffaloes

Affection	Number of animals	Percentage %
Thelitis	50	14.7
Obstruction	92	27.1
Wounds	48	14.1
Fistula	17	5
Ischemic necrosis	130	38.2
Papilloma	3	0.9
Total	340	100%

Table 2. The incidence of animals affected with ischemic necrosis in multiple teats

Teats number	Number of Animals	percentage	Teats
One	55	42.3	55
Two	40	30.8	80
Three	20	15.4	60
Four	15	11.5	60
Total	130	100%	255

Table 3. The sites of ischemic necrosis on teats

Site	Number	Percentage	Anterior teat	Posterior teat
Base	161	63.2	37	124
Middle	20	7.8	9	11
Base & middle	38	14.9	8	30
Whole teat	36	14.1	7	29
Total	255	100%	61	194

Table 4. The degree of tissues damage in teat by ischemic necrosis

Degree of damage	Teats number	Percentage
Theilitis with areas of ischemic necrosis	22	8.6
Superficial ischemic necrosis of teat skin	135	52.9
Deep ischemic necrosis of skin and intermediate	33	13
Full-thickness teat ischemic necrosis	13	5.1
Ischemic necrosis of the teat length except apex	38	14.9
Ischemic necrosis of the whole teat	14	5.5
Total	255	100%

DISCUSSION

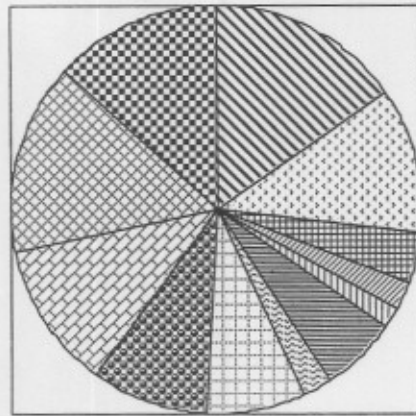
The surgical affections of the teats in buffaloes were previously discussed (3,4,5,7). An increase in the incidence of a specific form of teat necrosis in dairy buffalo was reported in this study. Therefore explanation of the observed condition was the aim of this study.

This disorder was recorded in dairy cattle under: teat sore by (8); summer sore, teat eczema and necrotic dermatitis (udder seborrhea) (1) and teat licking (9,10) while in buffalo was described under teat ulcer or sore and gangrene (3,4). Lately it was described under ischemic necrosis of the base of the teat in cattle and buffalo (11-14).

Ischemic necrosis was commonly observed in autumn and winter. This observation may be due to increase the incidence of calving and the damp conditions

at these seasons. Most of cases examined at summer were due to microfilaria. Similar observation was also noticed by (15,16).

The causes of this condition reported in dairy cattle could be self-licking of teat or injury due to flies, fly repellent spray, faulty milking machines and dipping with sodium-hypochlorite (9,10). Self-licking in buffalo is ruled out. The use of milking machines or teat dips was not practiced and fly repellents were not commonly used. Sore teats may occur as the result of wet milking, wet bedding and damp conditions in the stable in addition to rough handling of newly calved heifer by untrained milkers (17,18). Occurrence of the condition in the first weeks after calving especially in heifers, supposes that skin ischemia and necrosis may be due to excessive prepartum udder edema (1).



- Jan
- Feb
- Mar
- Apr
- May
- Jun
- Jul
- Aug
- Sep
- Oct
- Nov
- Dec

Fig 1. The incidence of animals affected with ischemic necrosis of teats during the year

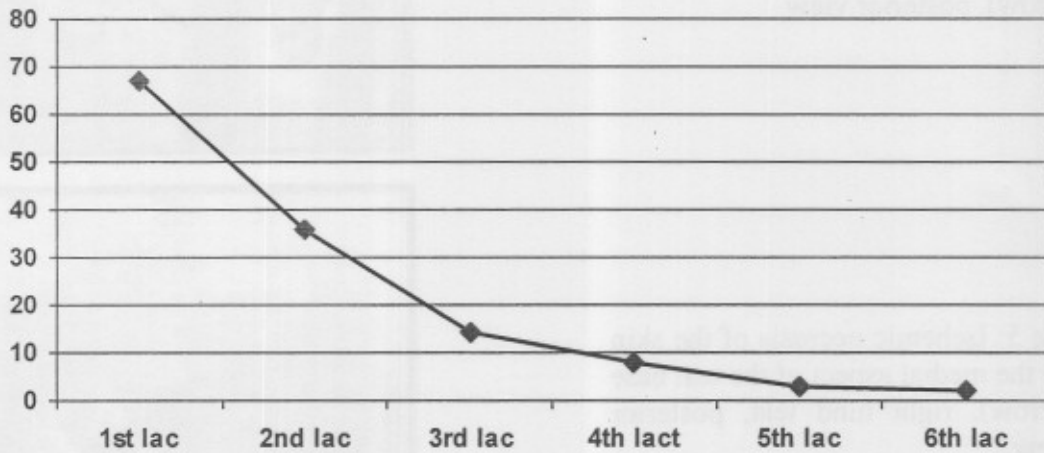


Fig. 2: The incidence of animals affected with ischemic necrosis of teats in relation to the lactation season

Fig 3: Enlarged edematous with overlying blackish dry rough thickened areas of skin (arrow) in the right hind teat, posterior view.

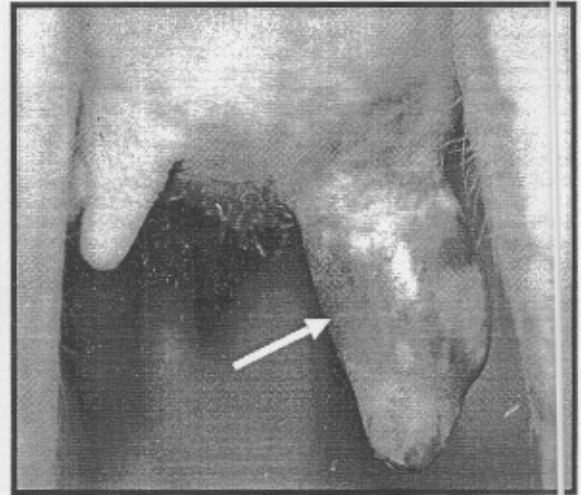


Fig. 4: Right hind chapped teat characterized by deep fissures (arrow), posterior view

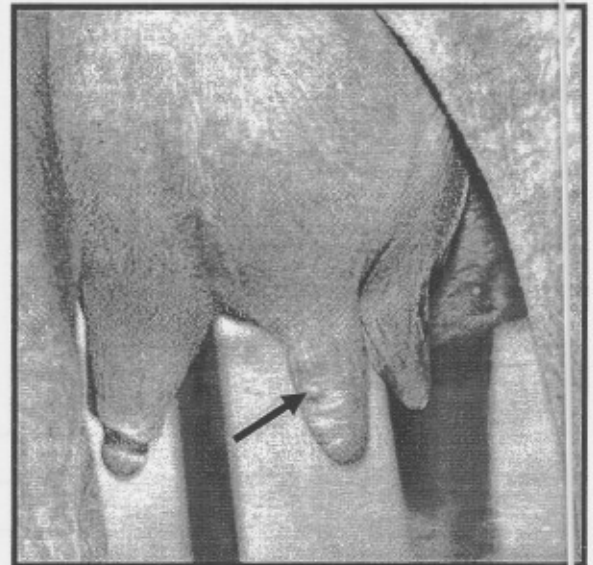


Fig 5: Ischemic necrosis of the skin on the medial aspect of the teat base (arrow), right hind teat, posterior view

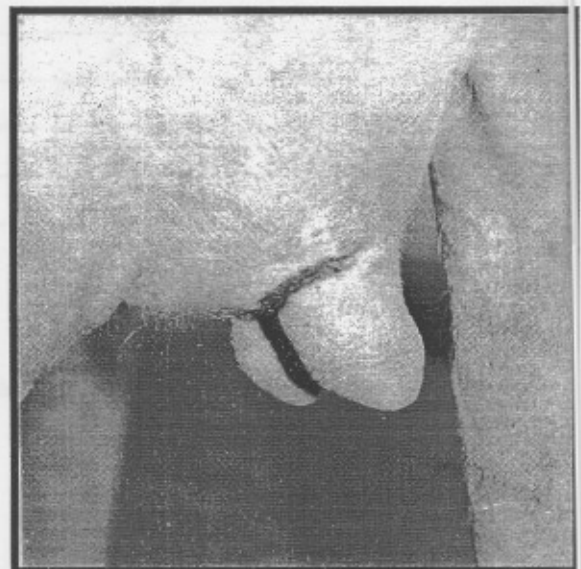


Fig 6: Ischemic necrosis surround the root of the left hind teat (arrow), side view

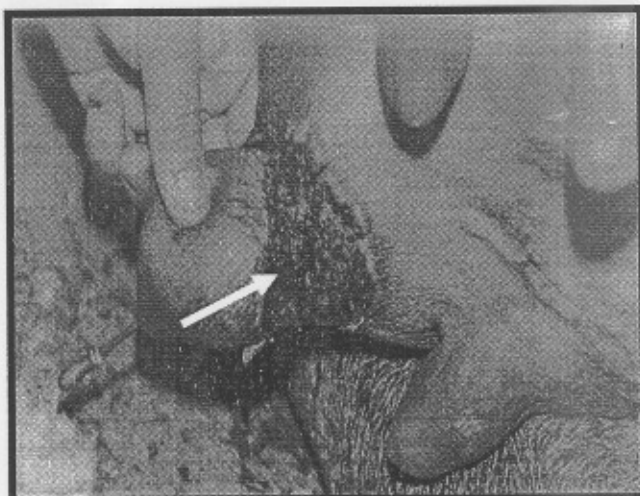


Fig 7: Deep ischemic necrosis of right hind teat characterized by thick brown scab cover which on removing leaving unhealed ulcer at its center and blood oozes from the lesion. Notice another ulcerative lesion on the hind limb (arrow), side view

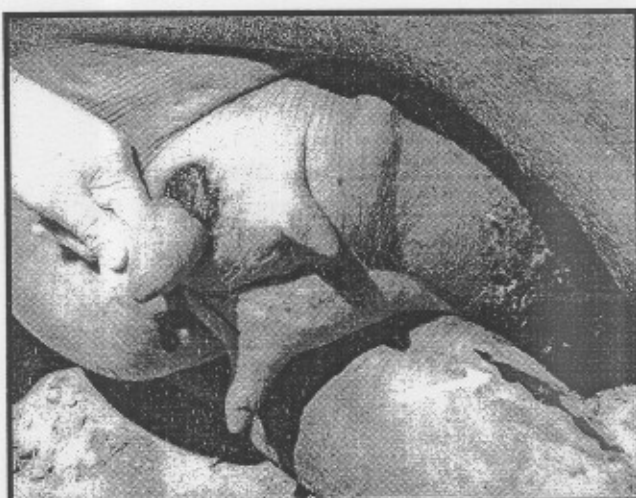


Fig 8: Ischemic necrosis at the four teats, side view

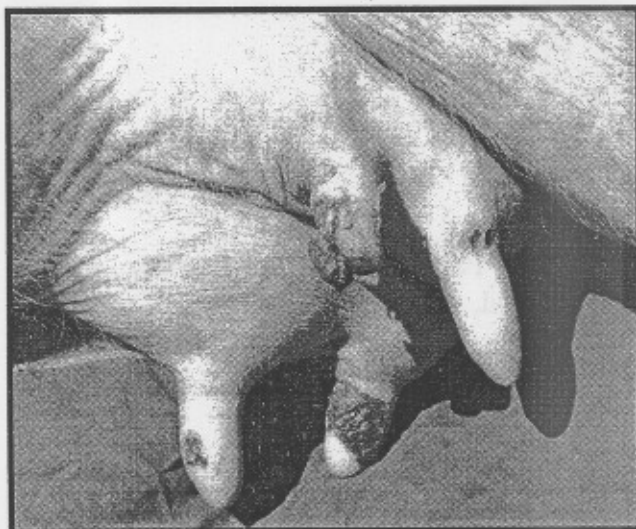


Fig 9a: Ischemic necrosis of full-thickness teat at its base, anterior left teat, side view

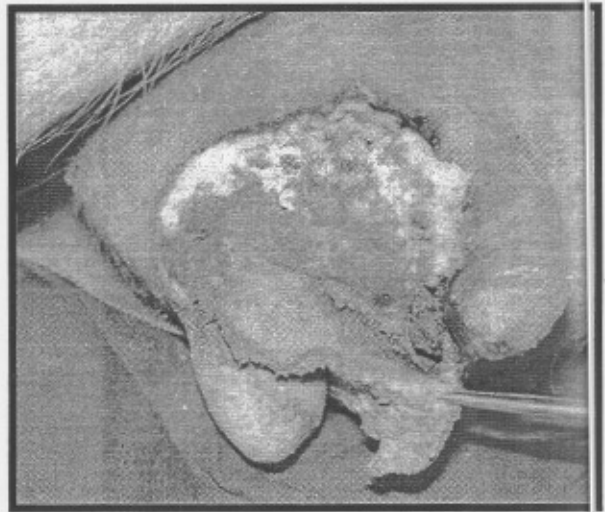


Fig. 9b: The wound observing the mammary tissue after removal of the necrosis, side view

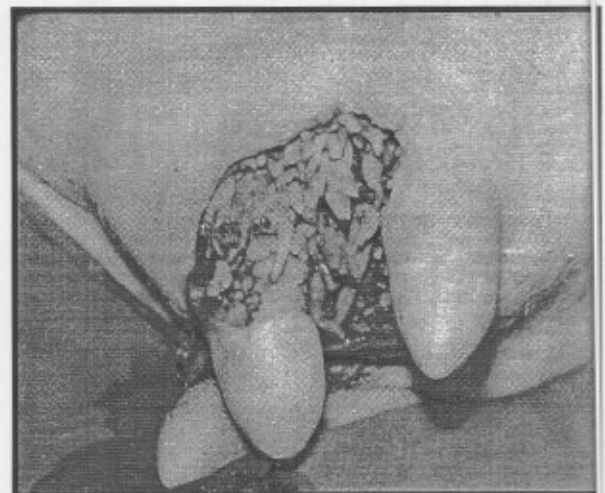


Fig 9c: Partial closure of the wound produced, side view

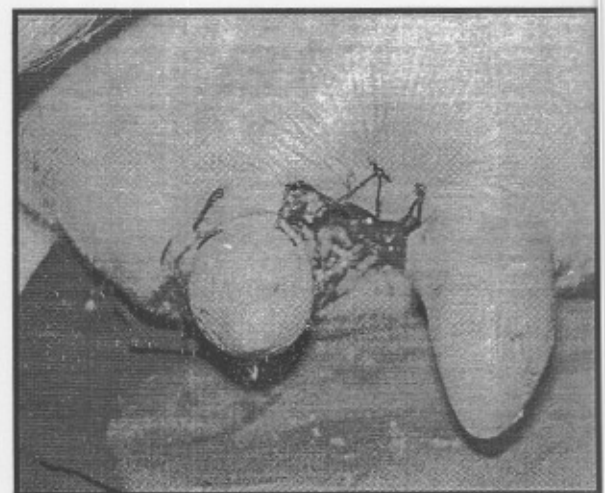


Fig.10a: Ischemic necrosis of full-thickness teat observing the teat sinus, left hind teat, side view



Fig 10b: The wound after massive debridement of unhealthy granulation tissue present in the site of ischemic necrosis, side view

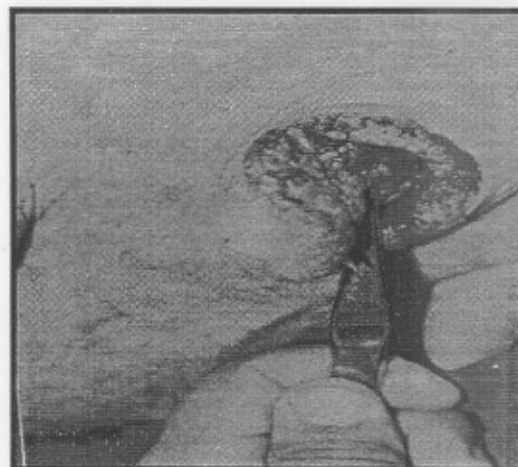


Fig 10c: Complete healing of teat with full-thickness necrosis by large scar (arrow), posterior view



Fig 11a: Ischemic necrosis included more than two third of teat length of the left anterior teat (arrow), side view

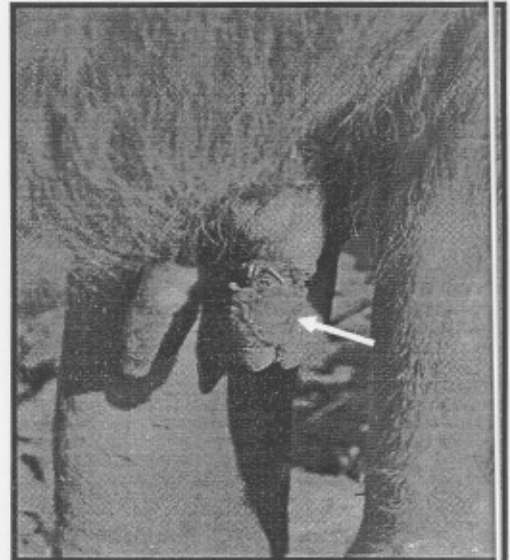


Fig 11b: Excision of the surrounding necrosis in teat with care to the teat sinus



Fig 11c: complete healing with contraction and epithelialization in two months, notice shortening of teat and scar constriction on left anterior teat, (arrow) posterior view

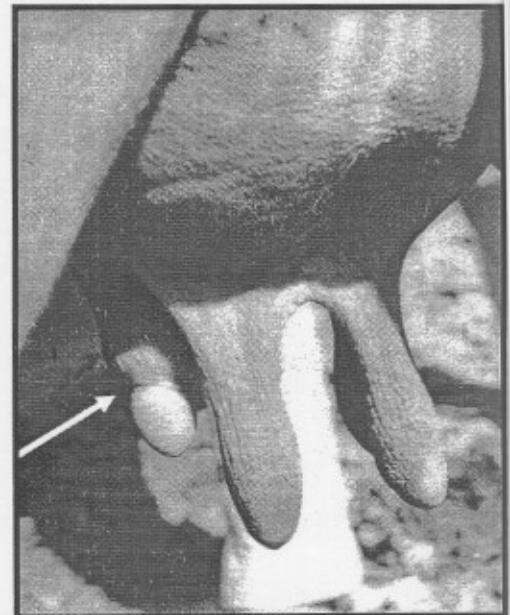




Fig 12a: Ischemic necrosis of the whole teat (gangrene)

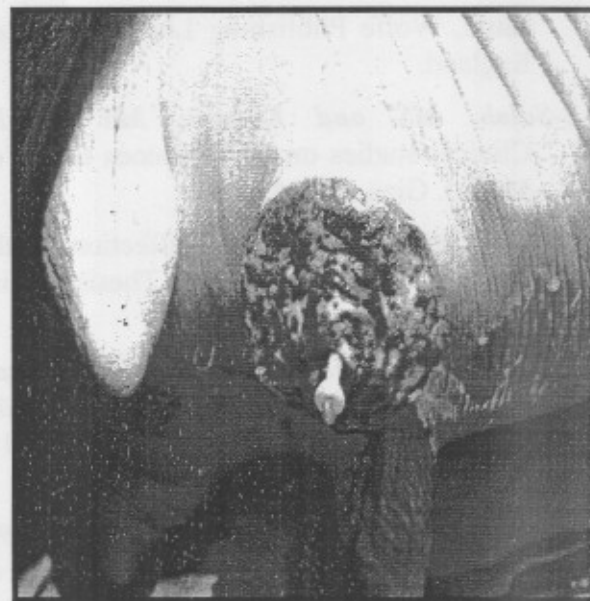


Fig 12b: The wound after amputation of gangrenous teat

The site of the initial lesion on the medial aspect of the base of teat is the point of impact of the mouth during suckling (11,17). However (11) reported the cause of this condition is still not known. It seems that the nature of the lesion is most likely to be associated with a disruption of highly vascular erectile tissue at teat base.

The early stage of inflammation of the teat and commonly observing the lesion at the base of the teat especially on its medial side in addition to the different stages of observation were described the progress of the condition. Recent investigators cited similar observations but they didn't clarify the different stages of their cases as recorded in this study (11-14). The initial changes are edema and thickening of the overlying skin which becomes necrotic and deep ulcerated. The necrosis may be extended to include half of the skin of teat or in some cases the whole teat.

Diagnosis was depended on the history, the clinical finding of lesion in addition microscopic examination. These methods were also used by (11-16).

The treatment ranged from local dressing and minimal debridement to massive debridement or amputation of teat. These methods were used according to the stage of ischemic necrosis diagnosed. Similar methods of treatment were also used separately in each case (13,19,20).

The methods of treatment needed to the different stages of the condition was effective for complete healing from 10-60 days according to the size of the lesion. Healing with or without minimal scar formation and normal milk flow was detected after treatment in the first three stages. Healing by contraction and impaired milk flow was noticed when ischemic necrosis included most of teat length. In cases when ischemic necrosis extended to the full-thickness or the whole teat, the sequel of treatment was teat fistulae and or drying off the corresponding quarter as the result of secondary infection and mastitis.

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الملخص العربي

تنقرز اسكيميا الحلمات في الجاموس وتأثيرها على الإنتاجية مستقبلاً

أحمد السيد بحيري

قسم الجراحة- كلية الطب البيطري- جامعة الزقازيق

وردت ثلاثمائة وأربعون جاموسة إلى قسم الجراحة- كلية الطب البيطري جامعة الزقازيق في الفترة من أكتوبر ٢٠٠٢ - يوليو ٢٠٠٧ بإصابات في الحلمات. وكانت نسبة الحيوانات المصابة بالتنقرز الاسكيمي ٣٨,٢%. وقد لوحظ أن نسبة الإصابة عالية بعد الولادة مباشرة من موسم الولادة. وتم فحص مائتان وخمسة وخمسون حلمة مصابة بالتنقرز الاسكيمي لوجود أكثر من حلمة مصابة في الحيوان الواحد. فكانت الناحية الداخلية من قاعدة الحلمات وخصوصا الحلمات الخلفية أكثرها مصابة. تم تقسيم هذه الإصابة إلى ستة أشكال مختلفة طبقاً لاتساع وعمق رقعة الإصابة. فكانت التغيرات الأولية عبارة عن تورم وتثخين أجزاء من جلد الحلمة التي سرعان ما تصبح متنقرزة و متقرحة. يمتد هذا التنقرز و يشمل جدار الحلمة وحتى أحياناً كل الحلمة. تم استخدام طرق عديدة لعلاج المراحل المختلفة لهذه الإصابة وتسجيل نتائج العلاج لكل مرحلة.