

# REAPPEARANCE OF LUMPY SKIN DISEASE (LSD) IN EGYPT

(With One Table and 11 Figures)

By

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عودة ظهور مرض الجلد العقدي في مصر

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أجريت هذه الدراسة خلال صيف وخريف عام ٢٠٠٥ م حيث ظهر مرض الجلد العقدي في مصر بصورة وبائية شملت عدة محافظات (الشرقية - الإسماعيلية - دمياط - الدقهلية - الغربية - بنى سويف - الفيوم - كفر الشيخ). للتعرف على بعض الجوانب الإكلينيكية والوبائية المتعلقة بالمرض في ٣ مزارع خاصة بالإضافة إلى ٢٠٠ من الحالات الفردية التي ظهرت عليها أعراض المرض في قرى تلك المحافظات. المزرعة الأولى تحتوى على ٨٣٣ بقرة فريزيان والثانية على ٢٠ بقرة فريزيان والثالثة على ٧٠ بقرة سويسرية. كانت نسبة الإصابة ٦٠,٠٢% في المزرعة الأولى وكانت ٥٠% في المزرعة الثانية بينما كانت ٤,٢٨% في المزرعة الثالثة وكانت نسبة الوفيات ٣% في المزرعة الأولى و ١٠% في المزرعة الثانية بينما لم تسجل أى حالة وفيات في المزرعة الثالثة. حدوث المرض في فصل الصيف يدل على الدور الهام للحشرات في إنتشار المرض. استمرارية حدوث المرض خلال فصل الخريف يدل أيضا على وجود وسائل أخرى لنقل المرض. تفاوتت الأعراض الإكلينيكية من حيوان إلى آخر وكانت الأعراض في مجملها عبارة عن ارتفاع في الحرارة، زيادة في إفراز اللعاب و امتناع الحيوان عن الأكل و تضخم في الغدد الليمفاوية السطحية مع ظهور عقد جلدية في معظم أجزاء الجسم. تطورت الأعراض إلى التهاب رئوى في بعض الحالات مما أدى إلى وفاتها. بإجراء التشريح المرضى لها تبين وجود عقد بالأجزاء العلوية من الجهاز التنفسى والرئة والجهاز الهضمى.

## SUMMARY

During late summer and autumn, 2005, outbreaks of Lumpy skin disease (LSD) were observed in different Egyptian governorates. So, this study was carried out to explore some epidemiological and clinical aspects of the disease in these recent outbreaks. Three private farms at Salheia and Gharbia governorate in addition to 200 individual animals belonging to farmers in different villages at different governorate were used in this

study. In the first farm out of the examined 833 Friesian cattle, 500 contracted the disease; 25 of them were died representing a morbidity rate 60.02%, mortality rate 3% and case fatality rate of 5%. In the second farm out of the examined 20 Friesian cattle, 10 contracted the disease; two of them were died representing a morbidity rate 50%, mortality rate 10% and case fatality rate of 20%. Whereas in the third farm out of the examined 70 Holstein cattle, 3 of them contracted the disease representing a morbidity rate of 4.28% whereas no fatality was recorded in this farm. The occurrence of the disease in late summer, 2005 indicating the role of biting insect in the disease transmission. The expansion of the outbreaks through autumn months suggest other modes of transmission rather than the insect as the needles that used in vaccination and medication in addition to the role of direct contact. Concerning the clinical findings that observed in these outbreaks, infected animals showed, pyrexia, anorexia, nasal discharge, salivation, depressions, external lymphadenopathy, dysgalactia, appearance of skin nodules of varying size which may cover the whole of the animal's body. These nodules might disappear spontaneously within 3 to 4 weeks or gathered to form large lumps that increase in the thickness of the skin at the coalesced lesions. Some lumps were opened and sloughed leaving ulcer in the detached area in some cases. Lesions are often found in the mouth and upper respiratory tract. Edema of legs, brisket, head regions and scrotum in males was observed. Lameness and recumbency was observed in cases with severe edema. It was complicated with respiratory manifestation, pneumonia, hemorrhagic enteritis, dehydration and later recumbency followed by death. Abortion was reported in one Holstein cattle in the fifth month. Postmortem findings observed in dead Friesian cattle were in the form of skin nodules in different part of skin; these nodules involve all skin thickness, subcutis and muscles in some cases, superficial lymph node enlargement. Similar nodules were observed in upper respiratory tract, lungs and digestive system

*Key words: Lumpy skin disease.*

## INTRODUCTION

Lumpy skin disease (LSD) is an infectious disease of cattle, occurs primarily in Africa and Madagascar and rarely in the Middle East. It is caused by a capripox virus that belongs to the family *Poxviridae* where the prototype strain of LSD is the Neethling virus (Alexander *et al.*, 1957; Tulman *et al.*, 2001 and 2002; Eeva, 2004 and Tuppurainen *et al.*, 2005).

Historically Lumpy skin disease (LSD) was first seen in Zambia (formerly Northern Rhodesia) in 1929 (Morris, 1931 and Haig, 1957). Initially, it was considered to be the result of hypersensitivity to insect bites. Between 1943 and 1945, cases occurred in Botswana (Bechuanaland), Zimbabwe (Southern Rhodesia) and the Republic of South Africa. The infectious nature of the disease was recognized at this time (Thomas and Mare, 1945; Von Backstrom, 1945; Diesel, 1949). LSD was identified in East Africa in Kenya in 1957 and in Sudan in 1972 and in West Africa in 1974, spreading into Somalia in 1983. From 1929 to 1986 the disease was restricted to countries in sub-Saharan Africa (Davies, 1981 and 1991). Recently affected countries include Kuwait in 1986-88 (Anonymous, 1988).

In May, 1988 LSD was first diagnosed clinically by Fayed in the Suez governorate, Egypt, where it was thought to have arrived at the local quarantine station with cattle imported from Somalia (Salem, 1989). The disease spread locally in the summer of 1988 to adjacent governorates; Ismaelia, Sharkia and apparently overwintered with little manifestation of clinical disease. The disease reappeared in summer, 1989 and within a period of 5 – 6 months, the disease spread to involve all the Egyptian governorates. LSD disease virus was isolated from Egyptian cattle in 1989 by House (1990). The disease was appeared in Israel during 1989 (Shimshony, 1989; Carn and Kitching, 1995 and Yeruham *et al.*, 1995), and in Sudan during 1989 – 1991 and in 1993 (Khalafalla *et al.*, 1993).

LSD is a subacute to acute cattle disease in Africa. It is characterized by extensive cutaneous lesions and signs typical of generalized poxvirus diseases (Davies, 1991; Carn, 1993 and Coetzer *et al.*, 1994). Arthropod-vectored transmission may be significant in epizootic outbreaks and in the spread of LSD into nonenzootic regions (MacOwen, 1959; Davies, 1991; Carn, 1993; Barnard *et al.*, 1994; Carn and Kitching, 1995; and Chihota *et al.*, 2001 and 2003). However, direct transmission of the virus by contact can play an important role in the disease spread (Yeruham *et al.*, 1995), where, the virus is excreted from clinically infected animals by lacrimation and nasal discharges (Weiss, 1968) and in the saliva (Haig, 1957). Attenuated lumpy skin disease virus strains and sheep pox virus have been successfully used as LSD vaccines in enzootic and outbreak areas; however, in areas free from both sheeppox and LSD, a safe and effective vaccine is required (Davies, 1991; Barnard *et al.*, 1994; Carn, *et al.*, 1994 and Yeruham *et al.*, 1994).

A presumptive diagnosis of LSD is based on clinical and epidemiological investigation and confirmation is achieved by animal inoculation, histopathological examination of the lesions, virus isolation and identification by electron microscopy or serology (Davies, 1991).

The disease is of economic importance in endemic areas where many cattle suffer severe emaciation, damage to hides, infertility in males and females, mastitis, loss of milk production and morbidity of up to 40%, although mortality rarely exceeds 3%. (Prozesky and Barnard, 1982; Davies, 1991; Carn, 1993; Barnard *et al.*, 1994 and Esposito and Fenner, 2001). Effective control of LSD requires accurate and rapid laboratory techniques to confirm a tentative clinical diagnosis (Tuppurainen *et al.*, 2005).

The aim of the present work was directed to throw on some epidemiological and clinical aspects on the recent outbreaks of LSD in Egypt and assessment of the role of sheeppox vaccine in the protection against the disease in the farms under study.

## **MATERIALS and METHODS**

### **Animals:**

During late summer and autumn, 2005, many cases of cattle at different Egyptian governorates suffered from appearance of skin nodules suspected to be infected with LSD. Two hundreds of cattle with different ages and sexes belonging to farmers (18 in Sharkia, 12 in Dakahlia, 22 in Demiat, 8 in El-Esmaelia, 4 in Beni Suef, 6 in Fayoum, 6 in Gharbia and 68 in Kafr El. Sheikh) were showing LSD lesions as well as 3 private farms at Salheia and Gharbia governorate were used in this study. Animals of the first farm were 833 Friesian cattle while those of the second farm were 20 Friesian cattle. They had been vaccinated with sheeppox vaccine 10 months before clinical manifestation of the disease. The third farm comprised 70 Holstein cattle. They had been vaccinated with sheeppox vaccine 7 months before disease occurrence.

### **Skin biopsies:**

Full skin thickness biopsies were taken under local anesthesia from recently formed skin nodules. Half of each biopsy was placed in 10% glycerol saline for virus isolation and the other half in 10% buffered formal saline solution for histopathological examination.

**Serum samples:**

Acute and convalescent serum samples were taken from each animal, kept at 20°C to be used in serological tests.

**Epidemiological investigation:**

Morbidity, mortality and case fatality rates were estimated according to Martin (1987).

**Clinical examination:**

All the studied animals were subjected to clinical examination according to Rosenberger (1979).

**Postmortem examination:**

Postmortem examination was done to 2 Friesian cattle according to Drurag and Wallington (1980).

**Histopathological examination:**

Histopathological examination was done according to the method described by Davies (1991), where, five microns thick paraffin section were prepared and stained with haematoxylin and eosin then examined microscopically according to Drurag and Wallington (1980).

**Virological examination:**

Virus isolation and serum neutralization test were done according to the method described by Alexander *et al.* (1957).

**Treatment trials:**

An intervention with usage of non steroidal anti-inflammatory in addition to antimicrobial agents (oxytetracycline with either sulphadimidine or tylosine tartarate) was recommended to the diseased animals. Animal's body was sprayed by equal volume of acetic acid 3% and lemon oil 0.1% as insect repellent.

## RESULTS

**Table 1:** Morbidity, mortality and case fatality rates in the examined animals.

	No. examined animals	No. diseased animals	No. dead animals	Morbidity rate	Mortality rate	Case fatality rate
1 <sup>st</sup> farm	833	500	25	60.02	3	5
2 <sup>nd</sup> farm	20	10	2	50	10	20
3 <sup>rd</sup> farm	70	3	0	4.28	0	0



**Fig. 1:** LSD nodules all over the body of infected cow.



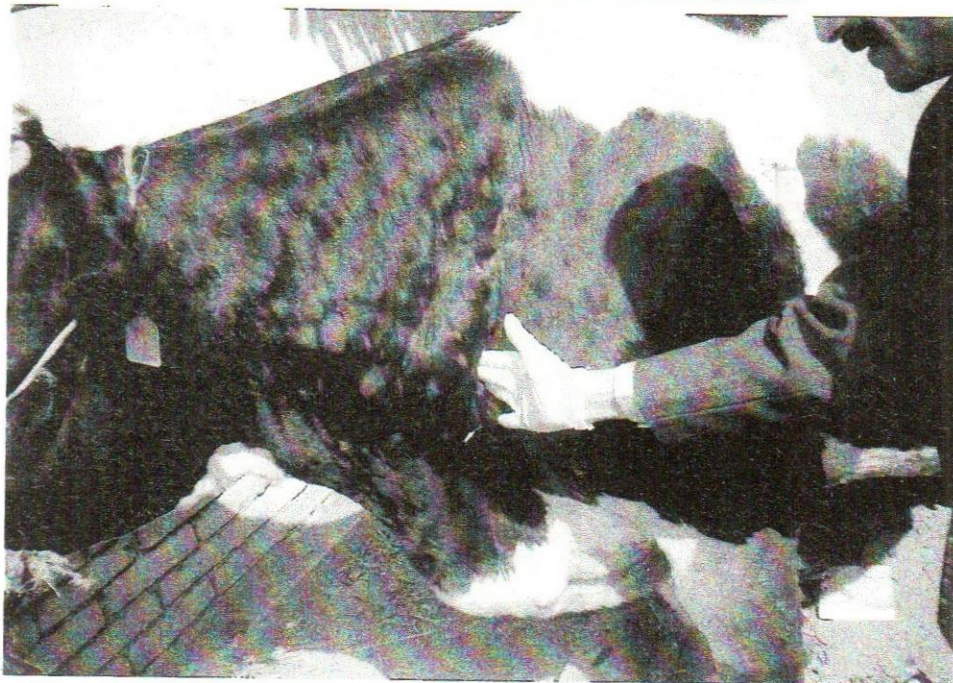
**Fig. 2:** LSD nodules on sides and back of infected cow.



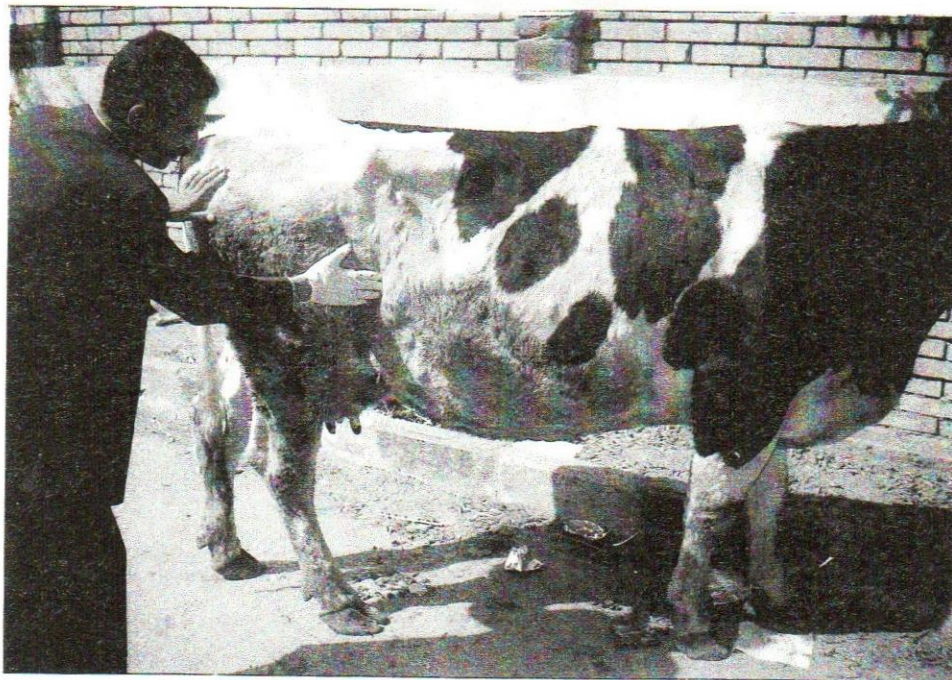
**Fig. 3:** Salivation, nasal discharges and nodules on the nostrils in LSD of infected cow.



**Fig. 4:** LSD nodules on the teats and udder of infected cow.

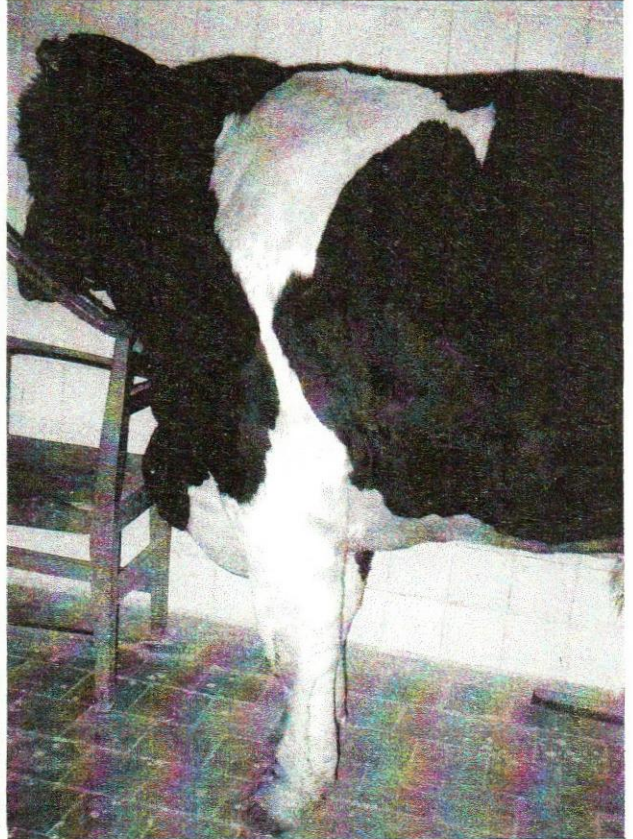
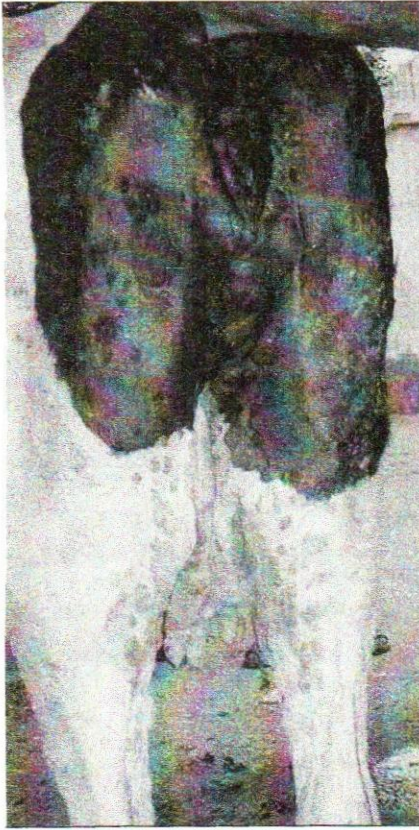


**Fig. 5:** Pre scapular lymph node enlargement and skin nodules in LSD of infected cow.

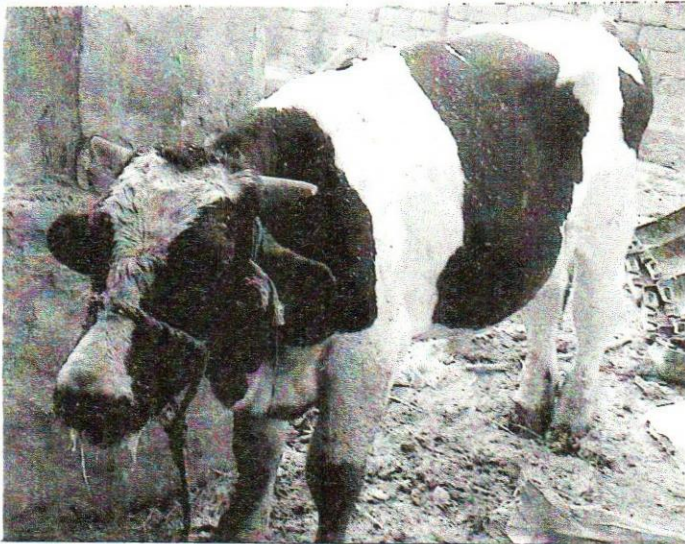


**Fig. 6:** Precrural lymph node enlargement in LSD of infected cow.

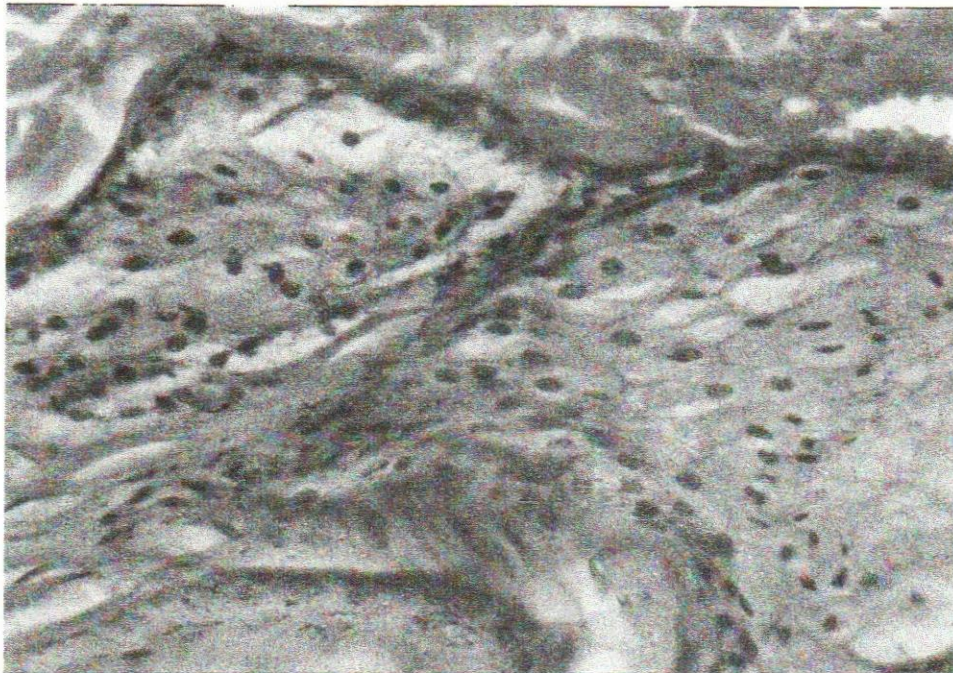




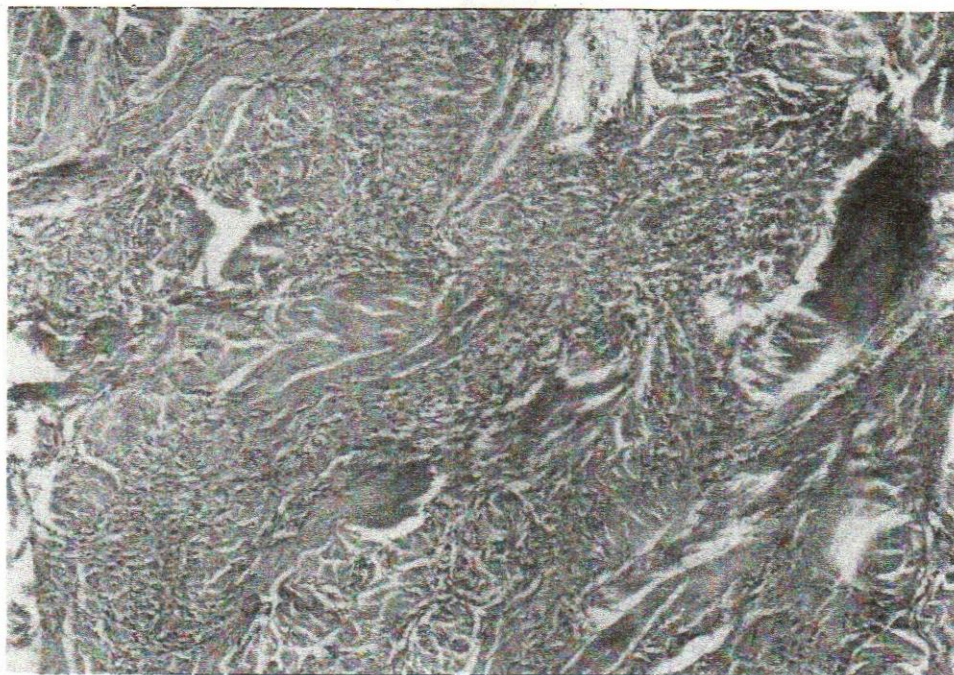
**Fig. 7:** LSD nodules on the udder and perineal region of infected cow. **Fig. 8:** Sternum and leg edema in LSD of infected cow.



**Fig. 9:** Depression, salivation, head and neck edema with extended head and neck in LSD of infected cow.



**Fig. 10:** Vacuolation, degeneration and intracytoplasmic eosinophilic inclusion bodies (H&E) x 400 in dermis of infected cow.



**Fig. 11:** Degenerative changes with leucocytic infiltration in dermis and hypodermis (H&E) x 100 in dermis of infected cow.

## DISCUSSIONS

Concerning the epidemiology of the disease in the examined 3 farms as shown in Table (1) it was observed that out of the examined 833 Friesian cattle in the first farm, 500 contracted the disease; 25 of them died representing 60.02% morbidity rate, 3% mortality rate and 5% case fatality rate. In the second farm out of the examined 20 Friesian cattle, 10 contracted the disease; two of them died representing 50% morbidity rate, 10% mortality rate and 20% case fatality rate. Lower rates were reported previously by Khalafalla *et al.* (1993) who reported in Friesian cattle a morbidity rate of 37.9% and a mortality rate of 4.2%. Higher rates were reported by Haig (1957) who recorded a high mortality rate in South Africa that reached 70% and attributed this to the virulence of the virus strain in that outbreak. Whereas in the third farm out of the examined 70 Holstein cattle 3 of them contracted the disease representing a morbidity rate of 4.28% whereas no fatality was recorded in this farm. In spite of the similarity in the degree of animal susceptibility in all farms, as they were all foreign breeds, it was noticed that the morbidity was higher, the signs were more severe and the fatality was recorded only in the first and second farms. This may be attributed to the longer time elapsed between vaccination and disease occurrence that lead to decline of protective immunity in animals of the first and second farms. This explanation agree with that of Capstick (1959); Davis (1982); Davis (1991) and Carn (1993) who found that CaPVs are serologically indistinguishable from each other and able to induce heterologous cross-protection. On the third farm, the onset of the disease was faced by rapid intervention by isolating the diseased animals with avoidance of contact with healthy ones as well as application of insect repellents. This minimized both the incidence and the severity of the clinical signs.

The disease was extended to involve many governorates where two hundreds of cattle of different ages and sexes belonging to farmers showed LSD lesions (18, 12, 22, 8, 4, 6, 62 and 68 cases in Sharkia, Dakahlia, Demiata, El-Esmaelia, Beni Suef, Fayoum, Gharbia and Kafr El-Sheikh respectively).

Concerning the epidemiological aspects of the disease it could be concluded that the disease was emerged again during late summer and autumn, 2005 as emerging disease. So, the reappearance of the disease in such large numbers producing local outbreaks in an endemic country like Egypt most probably due to lack of vaccination program.

At the farm level, the source of these outbreaks was not determined where no new animal was introduced to the examined farm. So this suggests the role of insect in disease dissemination from area to another. This is the second occurrence of LSD in Egypt since summer, 1988. LSD is thought to persist during cycles of subclinical infection in cattle or in old lesions (Davies, 1991). Moreover, wild ruminants have been found to be susceptible to the virus (Young *et al.*, 1970 and Greth *et al.*, 1992).

Occurrence of the disease in late summer and autumn, 2005 are similar to the previous observation of the disease in Israel on the same season on September, 1989. In addition, the first outbreak of LSD in Egypt also was recorded in summer, 1988. This related to the role of biting insect in the disease transmission (MacOwen, 1959; Davies, 1991; Carn, 1993; Barnard *et al.*, 1994; Carn and Kitching, 1995; Yeruham *et al.*, 1995 and Chihota *et al.*, 2001 and 2003). The expansion of the outbreaks through autumn months suggest other modes of transmission rather than the insect as the needles that used in vaccination and medication in addition to the role of direct contact.

Concerning the clinical findings that observed in these outbreaks as shown in Figures (1-9), infected animals showed, pyrexia (40°-42°C), anorexia, nasal discharge, salivation, depressions, external lymphadenopathy particularly prescapular and precrucial lymph nodes, dysgalactia, appearance of round, firm skin nodules slightly raised from the surrounding normal skin of varying size ranged from few millimeters to few centimeters, which may cover the whole of the animal's body and involving the whole thickness of the skin and subcutis. The hairs were erected over these nodules. The number of these nodules varies from few to several hundreds. These nodules might disappear spontaneously within 3 to 4 weeks or gathered to form large lumps that increase in the thickness of the skin at the coalesced lesions. Some lumps were opened and sloughed leaving ulcer in the detached area. In severe cases, lesions were found in the mucosa of the mouth and upper respiratory tract. Edema of legs, brisket, head regions and scrotum in males was observed. Lameness and recumbency was observed in cases with severe edema. This was complicated with respiratory manifestation to the extent of pneumonia, hemorrhagic enteritis, dehydration and later on recumbency followed by death in 25 cases in the first farm and two cases in the second farm in addition to 10 individual cases. Abortion was reported in one Holstein cattle in the fifth month. Similar findings were observed previously by Ali *et al.* (1990); Davies (1991); Abdalla and Gawad

(1992); Greth *et al.* (1992); Kalafalla *et al.* (1993) and Irons *et al.* (2005).

The variation in the clinical picture from the more severe generalized infection to the mild localized lesions may be immune dependant or related to the infective dose which transmitted either by blood sucking or tissue feeder insect Carn and Kitching (1995a).

Postmortem findings observed in dead Friesian cattle were in the form of superficial lymph node enlargement, skin nodules in different parts of skin. These nodules involve all skin thickness, subcutis and sometimes muscles. Similar nodules were observed in upper respiratory tract, lung and digestive system. Ulcerative lesions in the mucosa of respiratory and digestive tract, reddish hemorrhagic to whitish lesions in the lung, inter-lobular edema and nodules in the lung in addition to yellow fluids in the edematous swelling in the legs and different subcutaneous parts were observed. Similar changes were observed previously by Thomas and Mare (1945).

Histopathological appearance of intracytoplasmic inclusion bodies in addition to hydropic degeneration and necrosis in sebaceous glands and cells of hair follicles were the most prominent histopathological changes. Moreover, Ballooning degeneration of some of the epidermal cells is also visible (Figures 10 and 11). Similar lesions were recorded previously by Ali *et al.* (1990) and Yeruham *et al.* (1994).

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