AN OUTBREAK OF LUMPY SKIN DISEASE AMONG CATTLE IN UPPER EGYPT (EL-MENIA GOVERNORATE)

By

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SUMMARY

An outbreak of lumpy skin disease (LSD) was reported among cattle in El-Menia Governorate, Upper Egypt during the summer season of 1998. In July 1998, nine villages were visited at different localities of El-Menia Governorate and all suspected cases were clinically examined. The epidemiological data of the outbreak were also collected. Cattle of all ages were affected. The disease was observed in both indigenous and Holstein-Friesian cattle, with an increase in the severity of the disease among Friesian breeds. Epidemiological data revealed that the insect vectors, which present in high populations during the summer season, are incriminated in transmission and spreading of LSD virus. The morbidities were ranged from 20 to 75% in the nine investigated villages, while the mortality rates were estimated as less than 1%. Clinically the disease was characterized by fever, sudden appearance of firm rounded skin nodules, enlargement of superficial lymph nodes and edema of the limbs. The skin nodules were of about 1-4 cm in diameter and were covered with erected hair. LSD virus was detected by inoculation onto chrioallantoic membrane of embryonated egg and identified by indirect immunoflourscent technique. Transmission electron microscopy was used for demonstration of the LSD viral particles in the tissue samples and inoculated CAM. Also, the ultrastrucural features were described. Agar gel precipitation test (AGPT) and complement fixation test (CFT) were used for detection of LSD antibodies in the collected sera. The present study reported the re-occurrence of LSD epizootic in Egypt ten years after the appearance of the disease for the first time in the country in May 1988. Preparation of specific vaccine against LDS and application of an accurate vaccination programs are in need. Controlling of the insect vector will reduce the reappearance and spreading of the disease among cattle populations in the African countries.

INTRODUCTION

Lumpy skin disease (LSD) is a serious skin disease of cattle caused by a single strain of capripox virus closely allied to the viruses of sheep and goat pox and known as Neethling virus (Woods, 1988). LSD is caused by strains of capripoxvirus disease that are antigenically indistinguishable from strains causing sheep pox and goat pox (OIE Manual, 2000).

Lumpy skin disease virus strains isolated in Kenya over a period of some 20 years have proved to be serologically identical (Davies, 1982). Comparison of isolates of capripoxviruses collected from sub-Saharan Africa in sheep, goats and cattle by restriction endonuclease digestion of their purified DNA indicated that strains of capripoxvirus infecting cattle have remained very stable over a 30-year period and are closely related to strains recovered from sheep in Africa (Kitching et. al., 1989).

LSD virus was isolated for the first time from cattle in Egypt in two disease outbreaks in Suez and Ismailia Governorates (House et al. 1990). In Lower Egypt, Ali et. al. (1990) found that the incidence of LSD in 10 cattle and buffaloes herds from 6 different farms, between May 1988 and May 1989, was ranged from 21 to 71 %.

Epidemiologically, sheep pox, goat pox and lumpy skin disease differ, but all three viruses may be mechanically transmitted by biting insects (Carn, 1993). Transmission of LSD virus is thought to be predominantly by insects, natural contact transmission in the absence of insect vectors being inefficient (OIE Manual, 2000).

During LSD outbreaks in Egypt and Sudan, the most observable clinical manifestations were pyrexia (40-42°C) that persisted for 7 days, frequent nasal and lacrimal discharges and salivation. Appearance multiple dermal nodules, which occurred most frequently on the neck, brisket, back, thigh, leg, muzzle, perineum and udder. In severe infections, nodules were seen in mucous membranes of the mouth and nostrils. The superficial lymph nodes were swollen with thickness of the lymph vessels. Edema of the limbs and ventral abdominal wall were also seen (Ali and Obeid, 1977; Ali et. al. 1990; Agag et. al., 1992)

Laboratory confirmation of LSD is most rapid by the demonstration of typical capripox virions in biopsy material or desiccated crusts using the transmission electron microscope in combination with a clinical history of a generalized nodular skin disease and enlarged superficial lymph glands in cattle (OIE Manual, 2000). LSD virus could be grown on the chorio-allantoic membrane (CAM) of embryonated

hen's eggs. Maximum yield of the LSD virus were obtained in the CAM of 7-9 day embryos incubated at 33.5 and 35 °C for 5 to 6 days (Van Rooyen et. al. 1969).

A hallmark of the acute to subacute stages of the LSD lesions was the presence of intracytoplasmic eosinophilic inclusions in various cell types. The inclusions consisted of the viroplasm, which was identified as aggregates of electron-dense, finely granular to fibrillar deposits in which membrane-enclosed virions and occasional groups of tubular structures were observed (Prozesky and Barnard, 1982). In cell culture, Plowright and Witcomb (1959) found that LSD virus induced intracytoplasmic inclusions that differ than the inclusion bodies produced by sheep-pox and vaccina viruses.

Cattle were protected against challenge with rinderpest and lumpy skin disease viruses by vaccination with a recombinant capripoxvirus containing a full-length cDNA of the fusion protein gene of rinderpest virus (Romero, et. al., 1993) or containing the fusion protein (F) gene of rinderpest virus (Romero, et. al., 1994). In an experiment, the Romanian strain of sheep pox vaccine provided protection against generalized LSD in eight of ten cattle which were challenged with an Egyptian LSD field isolate (Michael et. al., 1996).

LSD was restricted to the sub-Saharan African countries until 1988 when the disease was appeared for the first time in Egypt (House, et. al., 1990). Since that time, several outbreaks were reported in different localities. The present study reported the occurrence of LSD outbreak in El-Menia Governorate, Upper Egypt in the summer season of 1998. The epidemiological data of the outbreak and clinical symptoms of the disease and ultrastructural features of the causative virus were described.

MATERIALS AND METHODS

During the investigation time, 110 serum samples as well as 18 tissue samples (table 1) were collected and submitted for laboratory diagnosis including transmission electron microscopy, egg inoculation, immuno-fluroscence, complement fixation test and agar gel precipitation test. Virological and serological tests were done at the Department of Virology, Animal Health Research Institute, Dokki, Cairo, while electron microscopic examinations were carried out at the pathology institute, Hanover veterinary school, Germany.

Samples

Tissue samples

Skin nodules or lumps were obtained aseptically from 18 animals with a typical clinical signs of lumpy skin disease. The prepared tissue samples were used for inoculation embryonated eggs onto the chrio-alantoic membranes, which subsequently submitted for indirect immunoflourscence and electron microscopic examination.

Serum samples

About 10 ml of whole blood were obtained from 110 LSD-clinically suspected cattle. For separation of the sera, the whole blood samples were centrifuged at 3000 rpm for 10 minutes. The clear sera were transferred into small tubes of 2 ml and kept at –20 C till the serological testing.

Table 1. Serum and tissue samples obtained from nine villages in El-Menia Governorate during LSD outbreak

Localities within El-Menia Governorate	Number of collected sera	Number of obtained tissue samples
El-Kaiat (El-Edwa city)	16	4
Dafash (Samalot city)	12	2
Dahmour (Magaga city)	10	2
El-Kafor (Matai city)	13	1
Al-Senaria (Bani-Mazar city)	14	2
Al-Nasria (Der-Mous city)	10	1
Darwa (Malawi city)	11	1
Abu-Kurkas (Abu-Kurkas city)	12	3
Nazla-Hussein (El-Menia city)	12	2
Total	110	18

Virus detection

Egg inoculation

The preparation of the collected tissue samples was carried out according to (Ali and Obeid, 1977). Fragments of the obtained skin nodules were minced in sterile sand and suspended in phosphate buffer saline (pH 7.4) containing 2000 units penicillin, 2000 µg streptomycin, 2000 µg neomycin sulphate and 50 units mycostatin per ml. The suspension was centrifuged and the supernatant used for egg inoculation.

Inoculation of embryonated hen eggs was done according to the technique described by Van Rooyen, et. al. (1969). 0.2 ml of the prepared suspension was inoculated on the chorio-allantoic membrane (CAM) of 9-12-day-old fertile chicken embryos. The inoculated eggs were incubated and candled daily; and those containing dead embryos were discarded. After incubation, the eggs were opened and the chorio-allantoic membranes were harvested.

Indirect immunofluorescent technique (IFT)

Impression smears of the infected chorio-allantoic membranes were fixed in acetone. The fixed smears were incubated with specific rabbit sera against the LSD virus and then stained with anti-rabbit conjugate according to the technique described by Davies, et. al. (1971).

Electron microscopy

The identity of the virus in the obtained tissue samples and inoculated chorioallantoic membranes was established by transmission electron microscopic examination based on the technique described by Munz and Owen (1966). Small blocks of the skin lesions and inoculated CAM were fixed in 5% sodium cacodylate buffered glutaradehyd and post fixed in 2% osmium tetroxide. Specimens were dehydrated in ascending grades of ethyl alcohol, passed through propylene oxide and embedded in Epon 812. Survey semithin sections were cut and stained with toulidine blue. Ultrathin sections were obtained from selected tissue blocks and stained with uranyl acetate and lead citrate and examined using Zeiss electron microscopy.

Serological tests

Both of the specific hyper-immune serum and the reference LSD virus were kindly supplied by the Abbassia institute for serum and vaccine production. Agar gel precipitation and complement fixation tests were used for detection of antibodies against LSD virus in the collected serum samples. The tested sera were firstly inactivated at 56 C for 30 minutes before serological examinations.

Agar gel precipitation test (AGPT)

AGPT was carried out against known infected-CAM antigen according to method described by Davies, et. al. (1971).

Complement fixation test (CFT)

In the CFT, serum samples were tested against known infected-CAM antigen according to Edwin (1969).

RESULTS

Epidemiology

The current study reported an outbreak of lumpy skin disease among cattle in El-Menia Governorate, Upper Egypt during the summer season of 1998, about ten years later than the first recorded outbreak of the disease in Egypt in May, 1988. Cattle of all ages were affected. The disease was observed in both indigenous and Holstein-Friesian cattle, with an increase in the severity of the disease among Friesian breeds. Insect vectors, which present in high populations during the summer season, are incriminated in transmission and spreading of LSD virus. The morbidities are ranged from 20 to 75% in the nine investigated villages, while the mortality rates are estimated as less than 1%.

Clinical symptoms

High fever of 40-41°C and sudden appearance of firm rounded skin nodules were the most observed clinical symptoms during this outbreak. Anorexia, dullness and depression were commonly noticed. Skin nodules or lumps were firm in consistency, rounded in shape, of about 1-4 cm in diameter and found on the neck, shoulder, thighs and back of the affected animal (figures 1, 2 and 3). These nodules were covered with erected hair. In some cases the skin nodules were found all over the body. Superficial lymph nodes, especially precrural and prescapular, were mainly enlarged. In severe cases, edema of the dewlap and one or more limb, which accompanied sometimes with lamness, was commonly observed. Weakness and emaciation were seen in most of the diseased cattle. The severity of the clinical manifestations was ranged from mild to moderate degree. Severe clinical cases of the disease, with nodules and lesions on the mucous membranes of the upper digestive and respiratory tracts, were not observed during this outbreak.

Laboratory diagnosis

Virus detection

Egg inoculation

Pock lesions were clearly observed on the CAM 4 days post inoculation.

Indirect immunofluorescent technique (IFT)

All impression smears (number 16) that prepared from the infected chorio-allantoic membranes were positive using indirect immunofluorescent technique (IFT).

Electron microscopy

Light microscopy of semithin sections revealed the presence of intracytoplasmic homogenes inclusion bodies in keratinocytes, endothelial cells, pericytes, and fibroblasts. Transmission electron microscopy revealed that the viruses developed in areas within the cytoplasm termed viroplasm (figure 4). These viroplasms contained developing virions and tubular structures (figure 5). Mature virions were observed in inside the intracytoplasmic inclusions as oval to rectangular particles with rounded corners (figure 6). A small electron dense granule could be seen within the core of the virus particles. Virus particles were surrounded by a multilayered envelope. The virus replication and formation of intracytoplasmic inclusions was associated with cytopathic changes including inter and intracellular edema, desmolysis of the intracellular prickles and finally cell rupture.

Serological tests

Agar gel precipitation test (AGPT)

72 (65.5%) out of 110 tested sera were showed precipitin lines in AGPT as shown in table (2)

Complement fixation test (CFT)

Table (2) shows also the results of CFT, where all of examined serum samples were positive in CFT with a titer ranged from 1/8 to 1/64.

Table (2): Detection of LDS antibodies in 110 collected sera using AGPT & CFT

No. of tested sera	AGPT Positive sera	CFT Titer of the positive sera			
		1/8	1/16	1/32	1/64
110	72	22	30	27	31

DISCUSSION

Lumpy skin disease is an infectious viral disease of cattle, which often occurs in epizootic form. It caused by LSD virus, which is member of family Poxviridae, genus Capripoxvirus and is antigenically indistinguishable from strains causing sheep pox and goat pox. Until 1988 The disease was confined to sub-Saharan Africa, but then spread into Egypt. Insect vectors are responsible for the transmission of the causative virus. Clinically, the disease is characterized by the formation of firm, circumscribed nodules in the skin and subcutaneous tissue. The skin nodules may cover the whole of the animal's body. The disease is considered of great economic importance due to damage of the hides, loss of production, and severe emaciation of the affected cattle (Mweene, et. al., 1996). The present study reported the re-occurrence of an outbreak of LSD among cattle in El-Menia Governorate, Upper Egypt during the period from May to August 1998.

The epidemiology of LSD is characterized by the apparent disappearance of the disease from the region for months or even years and then its sudden appearance with widespread outbreaks (Kitching and Carn, 1996). This epidemiological character could explain the reappearance of LSD among cattle in El-Menia Governorate in May 1998, ten years after the first outbreak of disease in Egypt, which occurred in May 1988.

The mode of transmission of the disease has not been clearly established. Contact infections do not readily occur and the evidence from the epizootiology strongly suggests that insect vectors are involved (Davies, 1991). Appearance of the disease in epizootic manner during the summer months, when a high population of insects are present and active, suggest also the transmission of LSD virus by the insect vectors.

In this study the most reported clinical symptoms of LSD in the affected cattle were fever of 40-41 C, which accompanied with anorexia, increase pulse and respiratory rate. Sudden appearance of firm rounded skin nodules of about 1-4 cm in diameter on the neck, shoulder, thighs and back of the animal. These nodules were covered with erected hair. In some cases the skin nodules were found all over the body. Superficial lymph nodes, especially precrural and prescapular, are mainly enlarged. In severe cases, edema of the dewlap and one or more limb, which

accompanied sometimes with lameness, is commonly observed. Weakness and emaciation were seen in most of the diseased cattle. These clinical manifestations agree with those reported previously in cattle during outbreaks of LSD in Egypt (Ali, et. al., 1990; Agag, et. al., 1992) in the Sudan (Ali and Obeid, 1977; Khalafalla, et. al. 1993), who found that the most characteristic clinical signs of LSD were pyrexia, nasal discharge, appearance of multiple skin nodules of varying sizes, and oedema of legs and brisket.

Systemic effects include pyrexia, anorexia, dysagalactia and pneumonia; lesions are often found in the mouth and upper respiratory tract in some cases of LSD. The severity of the disease varies considerably between breeds of cattle. Many cattle suffer severe emaciation and loss of production for several months. The skin lesions caused permanent damage to the hides (Davies, 1991). In the present study, the severity of the clinical manifestations was ranged from mild to moderate degree and severe cases of LSD with nodules on the mucous membranes of the upper digestive and respiratory tracts were not seen. Cattle of all ages were affected. The disease was more severe among cattle of foreign breeds. The morbidities were ranged from 20 to 75% in the nine investigated villages, while the mortality rates are estimated as less than 1%. Similar results were obtained previously by Ali and Obeid (1977) during an outbreak of LSD in the Sudan. Also, Outbreaks of lumpy skin disease in an imported Friesian and indigenous cattle were recorded in Khartoum State during the period of 1989-1991. The disease in purebred Friesian cattle was severe with a morbidity rate of 37.9% and a mortality rate of 4.2% while it was rather mild in indigenous cattle (Khalafalla, et. al. 1993).

Laboratory confirmation of LSD is most rapid by the demonstration of typical capripox virions in biopsy material or desiccated crusts using the transmission electron microscope (OIE Manual, 2000). During an outbreak of LSD in Sudan, the disease was diagnosed from clinical findings, isolation and identification of the virus and from electron microscopy (Khalafalla, et. al. 1993). Both indirect fluorescent antibody (IFA) and virus neutralization (VN) are useful in establishing a diagnosis of lumpy skin disease where clinical signs are evident (OIE Manual, 1992). In the current study, laboratory confirmation of the disease was depending upon detection of pock lesions on the inoculated CAM 4 days post infection. All impression smears, which prepared from the infected CAM, were positive by indirect immunofluorescent technique (IFT). LSD virus was inoculated by (Van Rooyen et al. 1969) onto CAM of embryonated eggs. "Pock" lesions in CAM were only appeared in 7-9 day embryos incubated at 33.5 and 35 °C for 5 to 6 days. Munz and Owen (1966) stated that LSD virus multiplies in the CAM of embrynoated eggs, but does not produce lesions similar to those caused by other poxviruses. LSD virus can be demonstrated in sections of CAM by staining with hematoxylin and eosin, by electron microscopy or immunofluorescence (OIE Manual, 1992).

In this study, light microscopy of semithin sections prepared from the skin nodules and inoculated CAM indicated the presence of intracytoplasmic homogenous inclusion bodies in keratinocytes, endothelial cells, pericytes, and fibroblasts. Experimentally, microscopic lesions in cattle infected with the lumpy skin disease virus comprised a granulomatous reaction in the dermis and hypodermis,

which extended to the surrounding tissue. During the early stages of the lesions a vasculitis and lymphangitis with concomitant thrombosis and infarction resulted in necrosis and oedema. Various cytopathogenic changes were observed in cells exhibiting viral proliferation. (Prozesky and Barnard, 1982). In cell culture Plowright and Witcomb (1959) found that LSD virus induced intracytoplasmic inclusions that differ than the inclusion bodies produced by sheep-pox and vaccina viruses.

In the present study, LSD virus and its inclusion bodies were demonstrated by transmission electron microscopy. The viruses developed within viroplasm. These viroplasms contained developing virions and tubular structures. Mature virions were observed in inside the intracytoplasmic inclusions as oval to rectangular particles with rounded corners. A small electron dense granule could be seen within the core of the virus particles. Virus particles were surrounded by a multilayered envelope. The virus replication and formation of intracytoplasmic inclusions was associated with cytopathic changes including inter and intracellular edema, desmolysis of the intracellular prickles and finally cell rupture. Those are similar to the results that previously observed by (Prozesky and Barnard, 1982), who demonstrated intracytoplasmic eosinophilic inclusions in various cell types during the acute to subacute stages of the lesions. The inclusions were consisted of the viroplasm, which was identified as aggregates of electron-dense, finely granular to fibrillar deposits in which membrane-enclosed virions and occasional groups of tubular structures were also detected. The use of the electron microscope as a diagnostic tool is becoming increasingly common (Davies et. al.1971). The introduction of the negative staining techniques has facilitated and considerably improved the electron microscopic demonstration of many viruses (Munz and Owen, 1966).

Monitoring of humoral antibodies, which was seen from day 2 of the onset of disease symptoms and lasted up to day 210, can be used as an effective tool for the diagnosis of LSD (Agag et. al., 1992). Kitching, et. al. (1986) had improved the sensitivity of the AGID test as a diagnostic test for capripoxvirus antibody detection. In the current study, AGPT and CFT were used for serodiagnosis of the disease, where 72 (65.5%) out of 110 tested sera were showed precipitin lines in AGPT and all of examined sera were positive in CFT with a titer ranged from 1/8 to 1/64.

The control of LSD without vaccination is extremely difficult in endemic areas. Recently developed live attenuated vaccines provide good, virtually lifelong, protection, which is dependent on stimulating cell-mediated immunity. Lumpy skin disease currently threatens to an extend beyond its existing boundaries, causing concern and renewed interest in vaccine development (Carn, 1993). The present study reported an outbreak of the LSD in El-Menia Governorate, Upper Egypt during the summer season of 1998. In conclusion, preparation of specific vaccine against LDS and application of well-controlled vaccination programs are in need. Controlling of the insect vector will reduce the reappearance and spreading of the disease among cattle populations in the African countries.

Aknowledgement
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REFERENCES

Agag, B.I.; Mousa, Sh.; Hassan, H.B.; Saber, M.S.; Nabila S. El-Deghidy and Aida M. Abd El-Aziz (1992): Clinical, serological and biochemical studies on lumpy skin disease. J. App. Anim. Res., 1: 13-23.

Ali, B.H. and Obeid, H.M. (1977): Investigation of the first outbreak of lumpy skin disease in the Sudan. Br. Vet. J. 133, 184-189.

Ali, A.A.; Esmat, M.; Attia, H.; Selim, A. and Abdel-Hamid, Y.M. (1990): Clinical and pathological studies on lumpy skin disease in Egypt. Vet. Rec. 127: 549-550.

Carn, V.M. (1993): Control of capripoxvirus infections. Vaccine (13): 1275-1279.

Davies, F.G. (1976): Characteristics of a virus causing a pox disease in sheep and goats in Kenya, with observation on the epidemiology and control. J. Hyg. (Lond), 76 (2): 163-171.

Davies, F.G. (1982): Observations on the epidemiology of lumpy skin disease in Kenya. J. Hyg. (Lond), 88 (1): 95-102.

Davies, F.G. (1991): Lumpy skin disease, an African capripox virus disease of cattle. Br. Vet. J. 147 (6): 489-503.

Davies, F.G.; Krauss, H.; Lund, J. and Taylor, M. (1971): The laboratory diagnosis of lumpy skin disease. Res. Vet. Sci., 12, 123-127.

Edwin, H.L. (1969): Complement fixation test modified kolmer microtechnique diagnostic procedure for viral and reckettsial diseases. 4th ed. 52-58 and 317-323.

House, J.A., Wilson, T.M.; El Nakashly, S.; Karim, I.A.; Ismail, I.; El Danaf, N.; Moussa, A.M. and Ayoub, N.N. (1990): The isolation of lumpy skin disease virus and bovine herpesvirus-4 from cattle in Egypt. J. Vet. Diagn. Invest , 2 (2): 111-115

Khalafalla, A.I., Gaffar Elamin, M.A. and Abbas, Z. (1993): Lumpy skin disease: observations on the recent outbreaks of the disease in the Sudan. Rev. Elev. Med. Vet. Pays. Trop. 46 (4): 548-550.

Kitching, R.P. and Carn, V. (1996): Lumpy skin disease. In: Manual of standards for diagnostic tests and vaccine: List A and B diseases of mammals, birds and bees, Paris, Office International des Epizooties (OIE), pp 93-101.

Kitching, R.P.; Hammond, J.M. and Black, D.N. (1986): Studies on the major common precipitating antigen of capripoxvirus. Journal of General Virology, Vol. 67, 139-148.

Kitching, R.P., Bhat, P.P. and Black, D.N. (1989): The characterization of African strains of capripoxvirus. Epidemiol. Infect., 102 (2): 335-443.

Michael, A.; Saber, M.S.; Soad, M. Sooliman; Mousa, A.A.; Salama, S.A.; Fayed, A.A.; Mounira, I. Nassar and house, J. (1996): Control of lumpy skin disease outbreak in Egypt with Romanian sheep pox vaccine. Assiut vet. Med. J., Vol 36, No. 71, 173-179.

Munz, E.K. and Owen, N.C. (1966): Electron microscopic studies on lumpy skin disease virus type "Neethling". Onderstepoort J. Vet. Res., 33 (1): 3-8.

Mweene, A.S.; Pandey, G.S.; Sinyangwe P.; Nambota, A.; Samui, K. and Kida, H. (1996): Viral diseases of livestock in Zambia. Jpn. J. Vet. Res. 44 (2): 89-105.

Office International des Epizooties (OIE) (1992): Lumpy skin disease. In: Manual of standards for diagnostic tests and vaccine: List A and B diseases of mammals, birds and bees. pp 57-61.

Office International des Epizooties (OIE) (2000): Lumpy skin disease. In: Manual of standards for diagnostic tests and vaccine: List A and B diseases of mammals, birds and bees. (http://www.oie.int/)

Plowright, W. and Witcomb, M.A. (1959): The growth in tissue cultures of a virus derived from lumpy skin disease of cattle. J. Path. Bact., Vol. 78, 397-410.

Prozesky, L. and Barnard, B.J. (1982): A study of the pathology of lumpy skin disease in cattle. Onderstepoort J. Vet. Res., 49 (3): 167-175.

Romero, C.H.; Barrett, T.; Evans, S.A.; Kitching, R.P.; Gershon, P.D.; Bostock, C. and Black, D.N. (1993): Single capripoxvirus recombinant vaccine for the protection of cattle against rinderpest and lumpy skin disease. Vaccine 11 (7): 737-742.

Romero, C.H.; Barrett, T.; Kitching, R.P.; Carn, V.M. and Black, D.N. (1994): Protection of cattle against rinderpest and lumpy skin disease with a recombinant capripoxvirus expressing the fusion protein gene of rinderpest virus. Vet. Rec. 135 (7): 152-154.

Van Rooyen, P.J.; Munz, E.K. and Weiss, K.E (1969): The optimal conditions for the multiplication of neethling-type lumpy skin disease virus in embryonated eggs. Onderstepoort J. Vet. Res., 36 (2): 165-174.

Woods, J.A. (1988): Lumpy skin disease-A review. Trop. Anim. Hlth. Prod. 20: 11-17.

Figure legends

Figure (1): 18-months old cattle with skin nodules on the neck, brisket and shoulders

Figure (2): Skin nodules covered with erected hair on the medial side of the thighs.

Figure (3): Skin nodules on the neck, shoulders and flank region of 5-years-old cow.

Figure (4): TEM showing viroplasm in a cell of the stratum spinosum.( ) Note intracellular edema (E), dilatation of rough endoplasmic reticulum (ER) and filamentous structure ( ). (Uranyl acetate and lead citrate, x11000).

Figure (5): Higher magnification showing virions in various stages of development. (Uranyl acetate and lead citrate, x22000).

Figure (6): TEM showing intracytoplasmic inclusion body in a keratinocyte. (Uranyl acetate and lead citrate, x11000).

Figure (7): Higher magnification showing the inclusion consisted of granular matrix in which virions were seen. (Uranyl acetate and lead citrate, x22000).

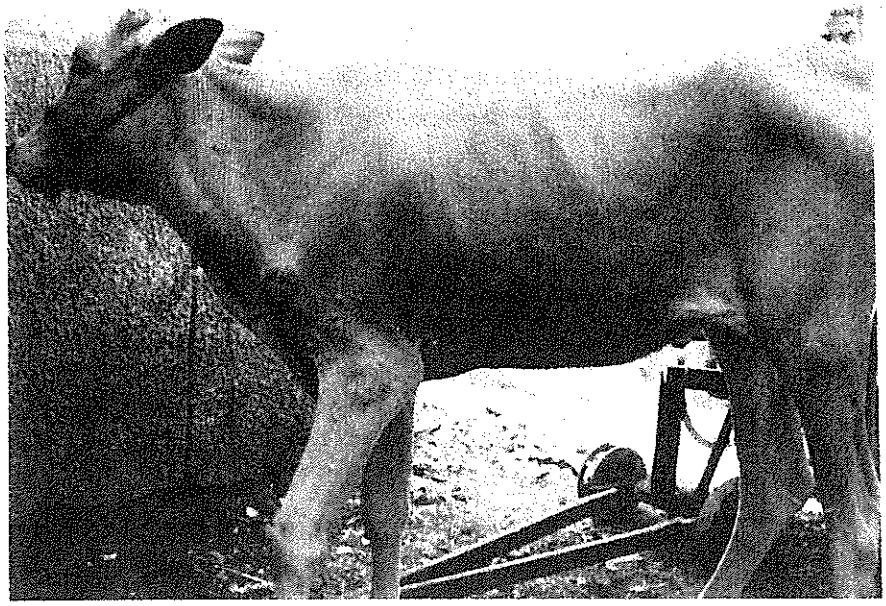


Figure 1.

Figure 2.

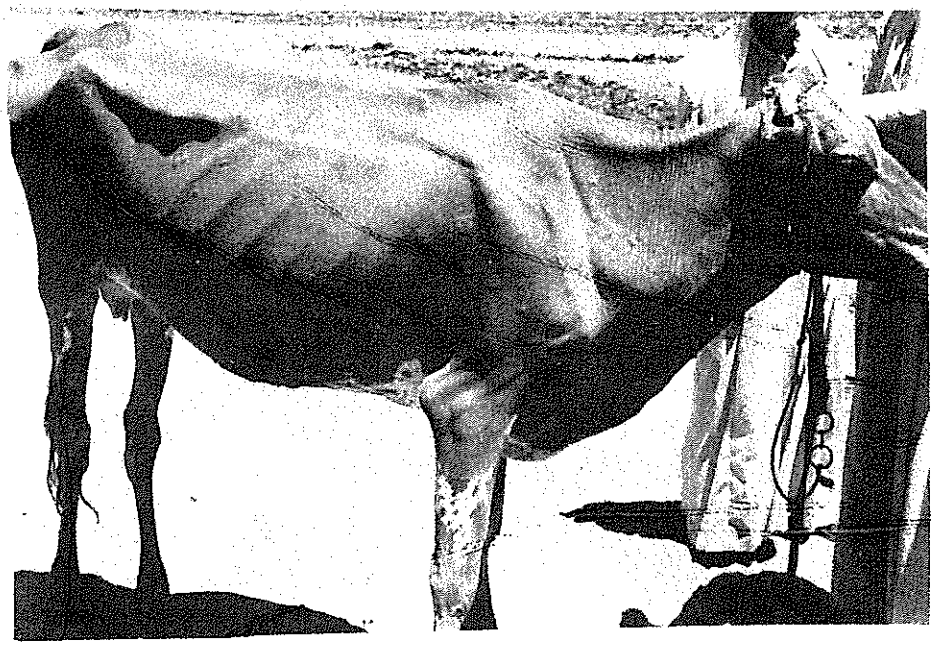


Figure 3

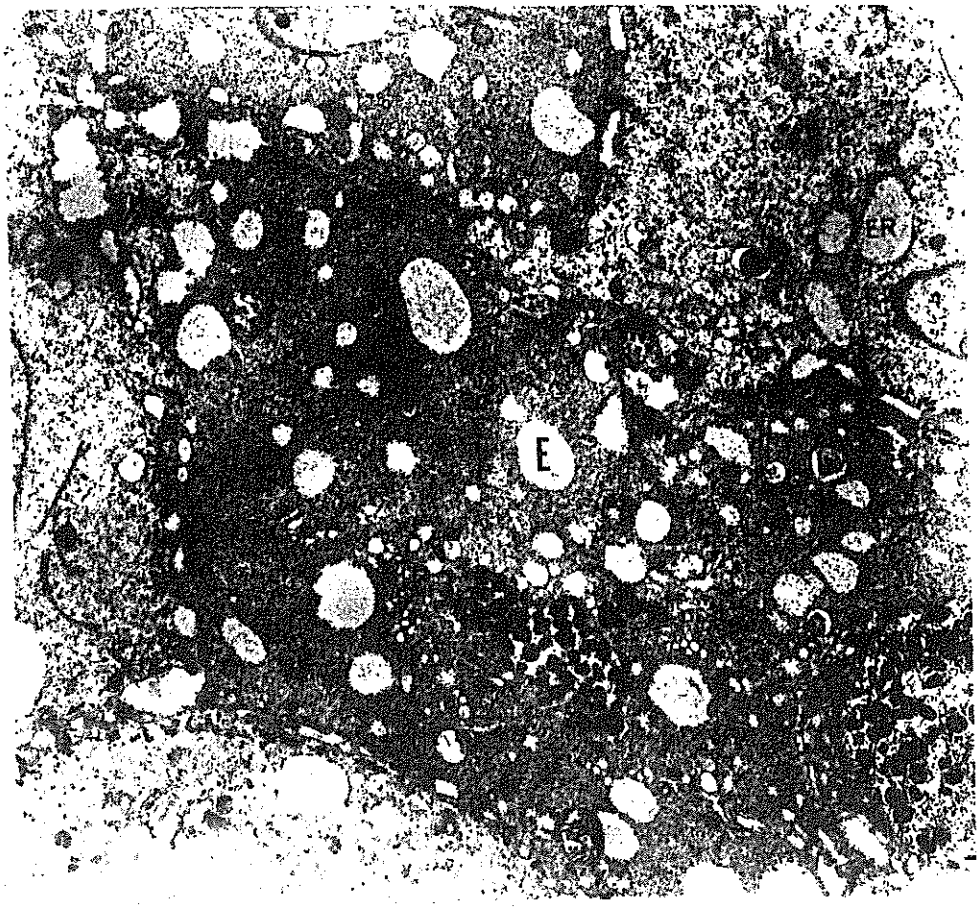
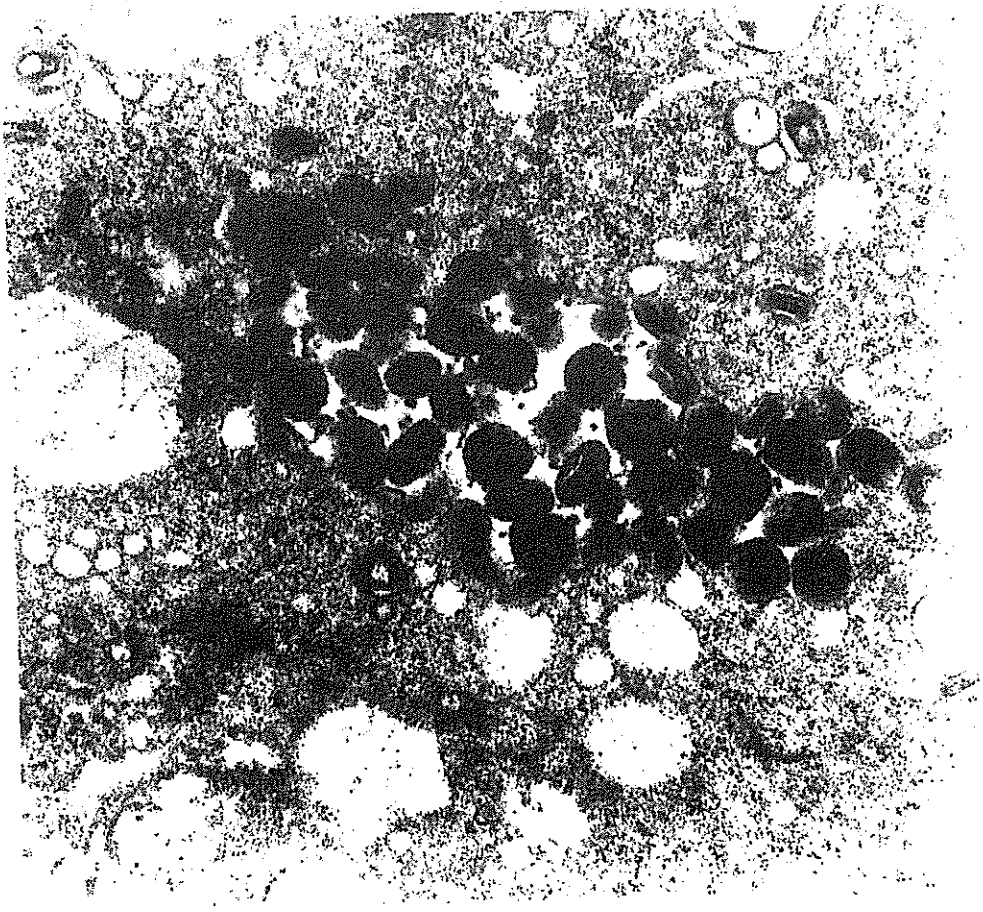


Figure (4)



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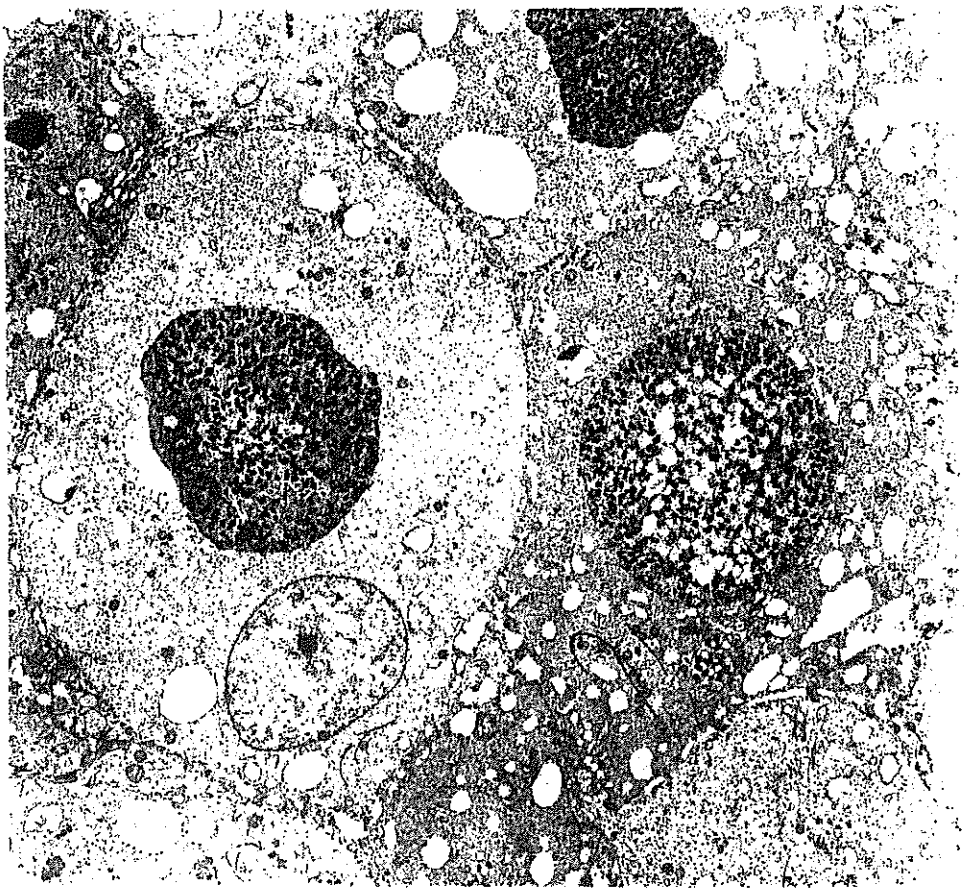

Figure (6)

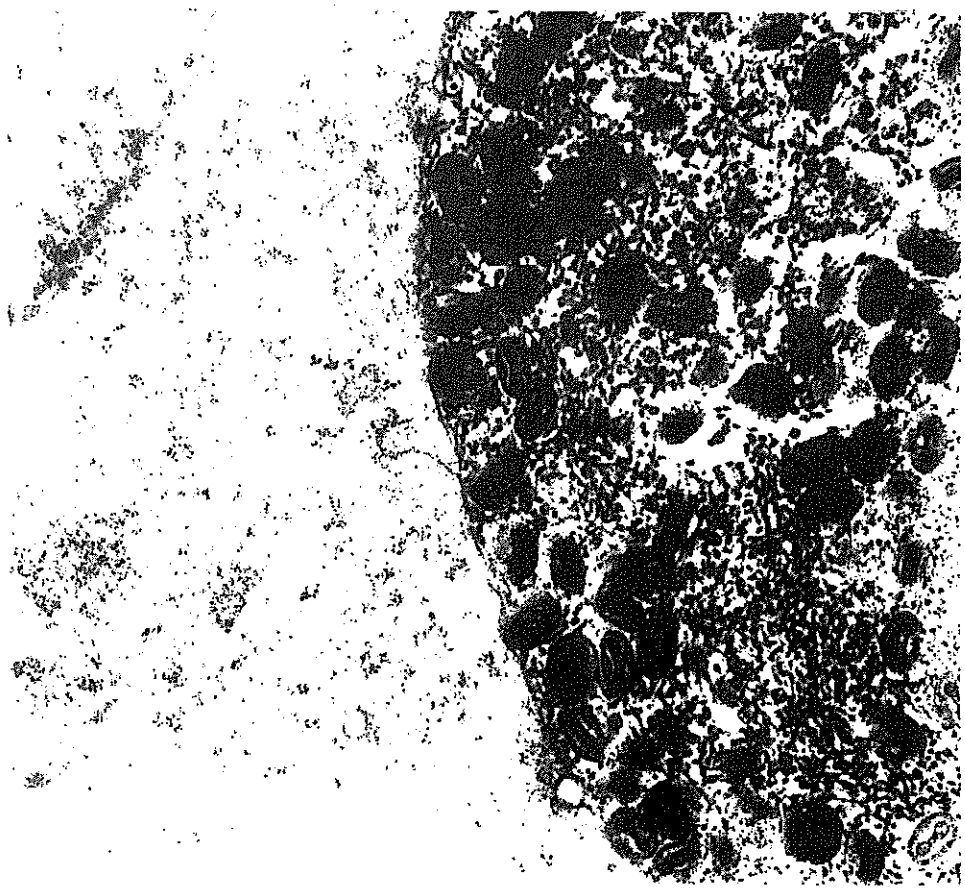

Figure (7):

الملخص العربى

وباء من مرض الجلد العقدي بين الأبقار بجنوب مصر (محافظة المنيا)

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سجلت الدراسة حدوث وباء من مرض الجلد العقدي بين الأبقار فى محافظة المنيا بجنوب مصر فى فصل الصيف لعام ١٩٩٨. خلال شهر يوليو ١٩٩٨ تم زيارة تسع قرى بأماكن متفرقة بمحافظة المنيا وتم الفحص الإكلينيكي للحيوانات المريضة. كما تم جمع المعلومات الوبائية الخاصه بالمرض. لوحظ إصابة الأبقار من مختلف الأعمار وكان المرض اكثر ضراوة بين الأبقار من السلالات الأجنبية (هولشتاين فريزيان) عنة فى الابقار من السلالات المحلية. اقترحت الدراسة الوبائية أن للحشرات الطائرة والمتواجدة بأعداد كبيرة خلال فصل الصيف دور فى نقل وانتشار المرض. تراوحت نسبة الإصابة ما بين ٢٠ الى ٧٥ فى المائة فى التسع قرى التى تم زيارتهم كما قدرت نسبة الوفيات بما لا يزيد عن واحد فى المائة. من الناحية الإكلينيكية تميز المرض بحمى وظهور مفاجى لعقد جلدية دائرية الشكل وتورم الغدد الليمفاوية السطحية مع انتفاخ الأرجل. وتراوح حجم العقد الجلدية ما بين ١-٤ سم فى القطر وكانت مغطاة بشعر منتصب. تم الكشف عن فيروس الجلد العقدى بواسطة الحقن فى البيض المخصب وتم التعرف علية باستخدام اختبار الفلورسنت المناعي المشع. وبإستخدام الميكروسكوب الإلكتروني النافذ تم الكشف عن ودراسة جزيئات الفيروس فى عينات الأنسجة المصابة وكذلك فى غشاء البيض المخصب المحقون. كما تم استحدام كلا من اختبار الترسيب فى الاجار واختبار التثبيت فى الكشف عن الأجسام المضادة للفيروس فى عينات السيرم. سجل هذا البحث تكرار حدوث وباء الجلد العقدى بعد حوالى عشر سنوات من اول ظهور لة فى مصر فى مايو من عام ١٩٨٨. أوضحت الدراسة ان هناك حاجة لتحضير لقاح خاص ضد فيروس الجلد العقدى وتطبيق برامج تحصين محكمة ضد هذا المرض. وأوصت الدراسة بالقضاء على الحشرات الناقلة للمرض الامر الذى سوف يقلل من تكرار حدوث هذا المرض فى قطعان الابقار بالأقطار الأفريقية.