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LUMPY SKIN DISEASE
I- CUTANEOUS AND TESTICULAR LESIONS
(With 18 Figs.)

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مرض الجلد العقدي
١ - التفغيرات الباثولوجية في الجلد والخصية

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لقد شخص وباء مرض الجلد العقدي حديثاً في جمهورية مصر العربية . وقد درست
الأعراض الإكلينيكية والصفة التشريحية وكذا التفغيرات الهستوباثولوجية في طبقات الجلد
المختلفة والخصية ووصفت النتائج كما نوقشت إمكانية تأثير فيروس المرض على خصوبة
ذكور الماشية .

SUMMARY

An outbreak of lumpy skin disease (LSD) in cattle was recently diagnosed in Egypt. Clinical signs and gross as well as histopathological alterations observed in the epidermis, hypodermis, subcutis and seminiferous tubules were described and illustrated. The pathogenesis was discussed, and the possibility of the testicular lesions as an etiologic factor for infertility in male animals was explained and interpreted.

INTRODUCTION

Lumpy skin disease (LSD) has been reviewed earlier in Africa by HENNING (1956) and HAIG (1957), and more recently by ALI and OBEIDI (1977) and DAVIES (1981). In the last two years, an outbreak of LSD among friezian and native bread cattle was recognized in Egypt. AGAG, *et al.* (1989) in collaboration with J.A. House in Plum Island, Animal Disease center, U.S.A. could confirm the diagnosis of the disease.

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Cutaneous lesions of LSD were described by many authors as epidermal proliferative and degenerative changes. Presence of eosinophilic intracytoplasmic inclusion bodies was considered diagnostic. In addition, erosive and ulcerative changes were also found in the respiratory and alimentary tracts (WEISS, 1968; BIDA, 1977; PROZESKY and BARNARD, 1982 and LOSES, 1986).

The concept of the pathological testicular changes in some viral diseases has been reported by JONES and HUNT (1983) and JUBB, et al. (1985). In LSD infection, the latter authors recorded an occasional finding of focal nodular orchitis in some affected animals. However, a detailed histopathological description of such testicular lesions or their pathogenetic mechanisms seemed to be dismissed.

The present investigation was employed to describe the testicular lesions in LSD, and to discuss their pathogenesis and significance. Furthermore, the dermatopathic effect of the virus for producing the epidermal and dermal lesions was studied.

MATERIAL and METHODS

Since October 1988, clinical signs as well as cutaneous lesions related to LSD were observed among cattle in Egypt. The affected animals were examined clinically. A wide range of tissue specimens from five necropsies, and thirteen cutaneous biopsies, excised under local anaesthesia, were taken and fixed in 10% neutral buffered formalin. The specimens were processed by conventional techniques for histopathological examination. Paraffin sections of 5 micron thick were stained with haematoxylin and eosin (H&E). Blood samples from the diseased and apparently healthy animals were also collected.

The present article only dealt with the cutaneous and testicular lesions.

RESULTS

Clinical observations:

During the febrile course of the disease, the affected animals showed excessive salivation, nasal discharge, lacrymation and inappetance. The morbidity rate was high and reached 73%, while the mortality rate was about 3%. Most of the affected animals revealed obvious cutaneous lesions on the entire body. Such lesions were expressed as greyish-white, circumscribed, flat-topped, firm nodules of 0.5 up to 4 cm. in diameter (Figs. 1,2). In the late stage of the disease, the cutaneous lesions had undergone necrosis and ulceration (Fig. 3). The ulcerated nodules appeared as raised lesions with circular core of mummified tissue surrounded by granulation tissue. Subcutaneous oedema of different parts of the body, and swelling of the superficial lymph nodes were also evident. In some cases, aggressive scrotal lesions associated with bilateral testicular swelling were noticed.

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Histopathological findings:**Cutaneous changes:**

The skin of the affected animals revealed various microscopical alterations in the epidermis, hypodermis, subcutis and adnexia. As regard to the epidermal changes, most of the examined skin autopsies and biopsies showed hyperplasia of the prickle cell layer with or without orthokeratotic and parakeratotic changes. The polygonal cells were swollen and exhibited cytoplasmic vacuolations (Fig. 4). In many areas, rupture of such cells with resultant circumscribed cavitations covered by thin epidermis were evident. Subepidermal clefts were oftenly seen. The microvesicles usually coalesced to form bullae or blebs (Fig. 5). In the vicinity of these lesions, disorganization of the granular cell layer was noticed. Coagulation necrosis associated with complete loss of the epidermal layers could be also observed (Fig. 6). Ruptured erosions as well as neutrophilic-infiltrated ulcers were found within or even above the epidermal surface (Fig. 7).

A peculiar and constant finding in all the examined cases was the presence of acidophilic intracytoplasmic inclusion bodies. The inclusions varied greatly in size and shape, usually homogenous and could be demonstrated in the slightly degenerated epithelium. In stratum spinosum, the inclusion bodies had the nuclear size or even larger. The cytoplasm of such cells appeared reticulated and their nuclei showed central chromatolysis (Fig. 8). Besides, invaginations with keratin-like material were recognized (Figs. 4,9). Some of these cystic structures contained proteinous acidophilic fibrillar material. The surrounding epidermal cells were flattened and manifested features of coagulation necrosis.

The extraepidermal changes were observed in the mesodermal structures of the stratum papillaris, reticularis and subcutis. Hair follicles and associated glands were also involved. Granulomas consisted mainly of centrally injured blood vessels surrounded by cellular infiltrates were commonly seen (Fig. 10). Histiocytes, lymphoid cells as well as degenerated polymorphnuclear leucocytes were predominant. The centrally located blood vessels and the other dermal vessels showed intimal proliferation and degeneration, medial fibrinoid necrosis, and thrombosis (Fig. 11). Coagulative necrosis of the surrounding mesodermal tissues was constantly noticed. The deep subcutis revealed diffuse interstitial dermatitis (Fig. 12). In some cases, the connective tissue elements and the muscle fibers showed necrosis and lysis (Fig. 13). Moreover, the sudoriferous glands had undergone compression atrophy with necrotic features. Occasional hyperplastic changes of the lining epithelium of some glandular ducts were also found.

Testicular changes:

The observed micromorphological alterations in the testis included degenerative and inflammatory changes in the seminiferous tubules and blood vessels. Such changes were bilaterally noticed and attained a diffuse manner. The seminiferous tubules were

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devoid of primary and secondary spermatocytes as well as spermatids. The spermatogonia and sertoli cells appeared resistant. Proteinous debris and multinucleated giant cells were found in some tubular lumina (Fig. 14). In addition, subacute focal interstitial orchitis manifested by hyperaemic blood vessels and mononuclear cell infiltrates was seen (Fig. 15). However, chronic fibrous interstitial reaction was not infrequently observed (Fig. 16). The small intertubular and the large interstitial blood vessels revealed intraluminal thrombosis, endothelial degeneration, medial necrosis and periarteritis (Figs. 17, 18).

DISCUSSION

On the basis of virological, serological and experimental animal infection, LSD was firstly diagnosed in Egypt by AGAG, et al. (1989). In the present investigation, clinical symptoms, gross and histopathological findings of the skin lesions confirmed the diagnosis (LOSES, 1986). Dermatitis nodularis seen by the authors, together with swelling of the superficial lymph nodes were also described by AYRE-SMITH (1960) and BIDA (1977).

It is imperically known that lesions produced by different viruses vary significantly according to their tropism to certain tissues. The vascular endothelium is infected in many viral diseases (ALEXANDER, et al. 1957 and PLOWRIGHT and FERRIS, 1959). The lesions in the vascular endothelium in such viral diseases are attributed to inclusion bodies or viral antigens (ALEXANDER, et al. 1957 and HOOK, et al. 1962). In our work, LSD virus showed direct cytopathic effect on the vascular endothelium of both skin and testis. Endvasculitis with characteristic proliferative and degenerative changes of the lining endothelium was found. It resulted in either luminal thrombosis and/or medial necrosis of the affected vessels. Hence, it could be concluded that LSD virus induced primary generalized angiopathic changes with subsequent alterations of the related tissues. PROZESKY and BARNARD (1982) correlated such endothelial damage either to the virus itself or the verions. In our opinion, vascular lesions of the vasa vasorum or vessel insudation may be responsible for such damage.

The epidermal changes ranged from intracellular oedema up to erosions and ulcerations. Similar findings were also described in both naturally and experimentally infected domestic and game animals (YOUNG, et al. 1970; NAWATHE and OSAGBA, 1977 and LOSES, 1986). The intracytoplasmic inclusions noticed in our work appeared to be diagnostic. Ultrastructurally, they were described as granular to fibrillar viral matrix. Besides, the peculiar intraepidermal cyst-like structures noticed in this article were similarly reported ultrastructurally by PROZESKY and BARNARD (1982) and described by CONROY and MEYER (1971) in swine pox as crystalloid deposits.

It is a matter of fact that the germinal testicular epithelium is highly sensitive to various adverse influences. In addition, testicular degeneration or atrophy is the

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most frequent cause of reduced fertility in male animals. In our study, severe degenerative changes accompanied with vascular disturbances were frequently observed. Such alterations could be attributed to either the vascular lesions and/or the febrile phase of the disease. The presence of fibrosing interstitial orchitis as a chronic lesion may be regarded as an additional factor for testicular degeneration.

Although no reports on the testicular lesions in LSD infection could be observed in the available literature, testicular changes were documented in malignant catarrhal fever and experimental blue tongue disease (JUBB, *et al.* 1985). The possible infertility of LSD virus may be transient as the regeneration of the germinal epithelium depends mainly upon the persistence of spermatogonia and sertoli cells. However, the long course of the disease and the presence of diffuse fibrous interstitial reaction of the testis may hinder the regenerative process. WIESS (1968) reported the occurrence of the LSD virus in the semen of affected animals for 22 days.

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DESCRIPTION OF FIGURES

- Figs. (1,2):** Animals showing cutaneous lesions.
- Fig. (3):** Cow showing advanced cutaneous lesions.
- Fig. (4):** Epidermis showing orthokeratitic and parakeratotic changes together with keratin invaginations and prikle cell vacuolations. H & E stain, X 250.
- Fig. (5):** Skin showing an intraepidermal bleb and subepidermal cleft. H&E stain, X400.
- Fig. (6):** Skin showing complete loss of the epidermal layers. H & E stain, X 250.
- Fig. (7):** Skin showing a neutrophilic infiltrated ulcer. H & E stain, X 250.
- Fig. (8):** Epidermis showing eosinophilic intracytoplasmic inclusion bodies in the prikle cells. H & E stain, X 1000.
- Fig. (9):** Epidermis showing keratin invaginations. H & E stain, X 250.
- Fig. (10):** Dermis showing a perivascular granuloma. H & E stain, X 250.
- Fig. (11):** Dermis showing angiopathic changes. H & E stain, X 250.
- Fig. (12):** Subcutis showing diffuse dermatitis, H & E stain, X 250.
- Fig. (13):** Subcutis showing coagulative necrosis. H & E stain, X 400.
- Fig. (14):** Testis showing degenerative changes. Notice the presence of giant cells. H & E stain, X 400.
- Fig. (15):** Testis showing subacute interstitial orchitis. H & E stain, X 160.
- Fig. (16):** Testis showing fibrous interstitial orchitis. H & E stain, X 250.
- Figs. (17,18):** Testicular blood vessels showing angiopathic changes. H & E stain, X 400.







