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CLINICAL AND PATHOLOGICAL STUDIES ON FOOT AND MOUTH DISEASE IN BUFFALOES

(With 7 Figs.)

By

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دراسات إكلينيكية وباثولوجية على مرض الحمى القلاعية فى عجول الجاموس المصري

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تناول هذا البحث ٢٢٧ رأس عجل جاموس من بين ٥٠٨٥ أعدت للتسمين في مزارع الصالحية بمحافظة الإسماعيلية . كانت المظاهر الخارجية بها مشابهة لمرض الحمى القلاعية مع وجود بثور وسحجات وقرح فى الفم خاصة الشفتين وظهر اللسان . وقد أعزى السبب لفيروس الحمى القلاعية عترة (٥) . وقد أظهرت الدراسة الهستوباثولوجية لهذه الحيوانات التهاب رئوى رشحى والتهاب كلوى وتنكرز فى إحداها والتهاب كبدى والتهاب فى عضلة القلب مع إرتشاحات وتوافد للخلايا الليمفاوية الصغيرة وقد صورت هذه الإصابات ووصفت ونوقشت تفصيلياً .

SUMMARY

In one fattening buffalo farm at Salhia, Ismailia Governorate 227 out of 5085 buffalo- calves showed clinical manifestations similar to Foot and Mouth Disease (FMD). The causative agent incriminated in this problem was FMD virus type "O". The virus caused oral lesions manifested as vesicles, erosions and ulcers inlips and dorsum of the tongue. The pathological studies revealed presence of bronchopneumonia, hepatitis, glomerulonephritis and myocarditis. These pathological lesions were illustrated and fully discussed.

INTRODUCTION

During the last 10 years, Foot and Mouth Disease (FMD) has taken an enzootic form in Egypt. The disease attacked susceptible animals, causing drastic losses of milk and meat and sometimes causing death of young animals. HUSSEIN, et al. (1975) and MOUSSA, et al. (1987) reported that FMD virus type "O" has been existed in Egypt. During the last epidemic in the period between 23 Feb. and 10 Jun. 1987, DAOUD, et al. (1988) reported high prevalence of FMD in different animal species (cattle, buffalo and shepp) in several provinces of Egypt. Nowadays, the importance and economic effects of FMD in Egypt receive proper consideration and become a disease of economic importance. So, application of active measures for its prevention and control is performed.

However, in a buffalo fattening farm, clinical manifestations similar to FMD appeared on a group of animals and we aimed in the present study to investigate the causative agent of these symptoms. Also, to record more informations ont he pathological picture of the disease and the histopathological changes inthe different organs and tissues of affected buffalo under our environmental conditions.

MATERIAL and METHODS

In a fattening buffalo farm at Salhia, Ismailia Governorate, certain clinical manifestations on buffalo - calves similar to the infection with Foot and Mouth Disease were observed.

Three calves were emergency slaughtered and subjected to postmortem examination. For histopathological studies, samples were taken from lips, tongue, lungs, liver, kidneys and heart and fixed in 10% neutral formalin. After proper fixation, tissues were embedded in paraffin, sectioned at 5-6 u thickness and stained with haematoxylin and eosin (H&E) and alcian blue stain (CLAYDEN, 1971).

For virological studies, samples from tongue epithelia, vesicles and mouth lesions of diseased cases were taken for isolation and identification of causative agent. Typing of FMD virus was carried out by complement fixation test (CFT) as described by Traub and MANOSA (1944).

RESULTS

1- Clinical findings:

The inspection of Salhia fattening buffalo farm at Ismailia Governorate revealed that the system of breeding is based on the periodical purchase of animals from different local markets and the problem began shortly after the introduction of purchased animals. From November 1988 to January 1989, 5085 buffalo - calves, 1 - 1.5 year-old, were collected from different governorates in Egypt (Sharkia, Gharbia, Assiut) to be fattened in the farm. At this period, 227 buffalo - calves were isolated with clinical manifestations characterized by: rise of temperature, anoxia, severe salivation, emaciation, lameness and appearance of vesicles on gum and dental pad as well as erosions on the dorsum of tongue (Fig. 1, 2). The morbidity rate reached 4.46% without mortality except the three cases that were emergency slaughtered for the subsequent examinations.

2- Gross pathology:

Three buffalo - calves were subjected to postmortem examinations. The carcasses were markedly emaciated. Vesicles about 0.5 cm in diameter contained viscid fluid and erosions were seen on upper and lower lips as well as on the dorsum of the tongue (Fig. 1, 2).

The heart:

In all cases, it was flabby and the coronary groove was surrounded with gelatenous materials and presence of petechial haemorrhages especially on epicardium, endocardium and muscular papillae.

The lungs:

In three cases, the lungs were congested with the presence of small irregular patches of consolidation. The cut surface along bronchi, bronchioles and lung tisues revealed the presence of slimy fluid.

The livers:

Were moderately enlarged in size, pale yellowish to gray incolour, friable in consistency. Its cut surface pulged. The gall bladder was distended with bile.

The kidneys:

In most examined cases, the kidneys were congested, the perirenal fat appeared gelatinous. In one case, small triangular erea about two mm in diameter was seen in cortical tissues. It was gray red in colour, sharply demonstrated from the surrounding tissues and its cut surface was tough.

3- Laboratory diagnosis:

The complement fixation test revealed that in all cases, the causative agent was Foot and Mouth Disease virus type "O".

4- Histopathological findings:

Lips and tongue showed in the epidermal layer degenerative changes in the epithelium of stratum spinosum. The cells become swollen, sometimes detached from each other and their nuclei were pyknotic. Small vesicles were seen under the stratum corneum and lucidum and formed as a result of liquefactive necrosis, oedema, mononuclear cellular infiltration (Fig. 3). The vesicles were coalesced leading in some areas to complete necrosis of stratum granulosum and spinosum which become dteached and separated from the stratum basalis (Fig. 4). The stratum basalis was destroyed in some areas leading to ulcer formation. The ulcerative areas were infiltrated by mononuclear cells and few neutrophils (Fig. 5). The dermis showed marked haemorrhages and infiltration by mononuclear cells.

The lungs:

Bronchi and bronchioles revealed plugs of inflammatory exudate intermixed with desquamated epithelial cells and small round cells in their lumina. Most of blood vessels (peribronchial, peribronchiolar and interalveolar) were dilated and engorged with blood. Perivascular lymphocytic cellular aggregation were seen specially into bronchial wall. Small bronchioles showed hyperplasia of their epithelial lining, leading to partial obliteration of their lumen. Marked thickening of alveolar wall in some areas was due to haemor-

rhages nad congestion of thier cfapillaries as well as infiltration with small round cells. Spots of alveolar emphysema or even rupture of its wall wall were seen.

The liver:

Most of hepatocytes showing a picture of cloudy swelling. Activation of Von Kupffer's cells especially perivascular was seen. The blood vessels especially central and portal veins as well as sinusoids were congested. Mononuclear cellular infiltration was seen in the portal triads beside connective tissue proliferation.

The kidneys:

Showed congestion in some sporadic areas, manifested by dilation and engorgement of blood bessels with blood. Some cortical tubules were showing vascular degeneration manifested by pale staining cytoplasm, containing well defined vacuoles. Fine granular, homogenous acidophilic materials replacing destroyed tubules were seen. Focal areas of proliferativeinflammation were present, which were manifested by diffuse cellualr infiltrations and proliferations mainly fibroblasts, fibrocytes, few lymphocytes and plasma cells. In one case, the kidneys showed focal triangular areas of necrotic tissues, represented by fragmented cells nad pyknotic nuclei. The affected areas were sharply demarkated from the surrounding healthy tissues by a zone of cellular infiltration mostly small round cells and fibroblast cells.

The heart:

Showed slight congestion nad haemorrhages (Fig. 6). Degeneration of some cardiac muscle fibres was seen. Focal spots of small round cells infiltration were prominent in most cases (Fig. 7).

DISCUSSION

Since the last outbreak occurred in 1987 among cattle, buffalo and sheep in different governorates in Egypt, restrict vaccination programe has been adopted allover the country. However, the present studies revealed the infection of 227 out of 5085 animals in a fattening buffalo farm with FMD virus type "O". This type of virus has been existed in Egypt for several years (DAOUD, et al. 1988). The appearance of the FMD among these animals indicates that these animals were not properly vaccinated. Perhaps, the system of breeding; dependent on the periodical purchase of buffalo - calves from different local markets, is responsible for the lack in strictly following the instruction of proper vaccination against FMD.

The clinical picture of the disease as well as the macroscopic lesions observed in the different organs of infected buffaloes were similar to those commonly observed in cattle (BLOOD, et ai. 1983; DUTTA, et al. 1983 and MOUSSA, 1985). The signs were directly related to the lesions of the disease. The presence of oral lesions in the oral mucosa, produces excess salivation and makes eating painful, thus the infected animals refuse food and water. These animals may remain as carriers. JUBB, et al. (1985) ment-

ioned that the carrier state persists for up to 6 months, probably longer, and this fact is of obvious epidemiological significance.

The histopathologic lesions developed in the different organs and tissues of infected buffaloes revealed the formation of vesicles, erosions and ulcers in lips and dorsum of tongue. The examination of internal organs revealed that the buffaloes were suffered from bronchopneumonia, hepatitis, glomerulonephritis, infarction and mild myocarditis. The main pathologic features in these organs were cellular degeneration and necrosis, congestion, haemorrhages and mononuclear cellular infiltration. These lesions were commonly seen in Foot and Mouth Disease and coincided with those reported by JONES and HUNT (1983); MARTIN (1983) and JUBB, et al. (1985). The authors reported that the primary lesions were developed in the oral mucosa and the virus soon gains access to the blood stream produced secondary lesions in the epithelial tissues.

The cardiac lesions observed in buffalo - calves were similar to those reported by KHATER (1966) in cattle. It was suggested that the myocarditis aphthosa as a constant finding in FMD is the result of intensified virus multiplication in the heart muscle. The author mentioned that the virus in young animals caused extensive coagulative necrosis while in mature ones, it produced the more typical epithelial reaction with intense lymphocytic infiltration. This corresponds to the findings of PLATT (1956, 1958) and SEIBOLD (1960) in their studies on mice and guinea pigs experimentally infected with FMD virus.

In conclusion, Foot and Mouth Disease in Egypt still requires the application of active measures of its prevention and control. Moreover, our breeding farms must be based on progeny of selected dams. The offsprings may be used for fattening proces. This system will perform the strictly following the instructionof proper vaccination against FMD and other diseases.

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LIST OF FIGURES

Fig. (1): Dental pad showing erosion about 1 cm in diameter.

Fig. (2): Dorsum of the tongue showing irregular areas of erosions.

Fig. (3): Toggue showing small vesicle under the stratum cornium and lucidum H & E. Stain X 160.

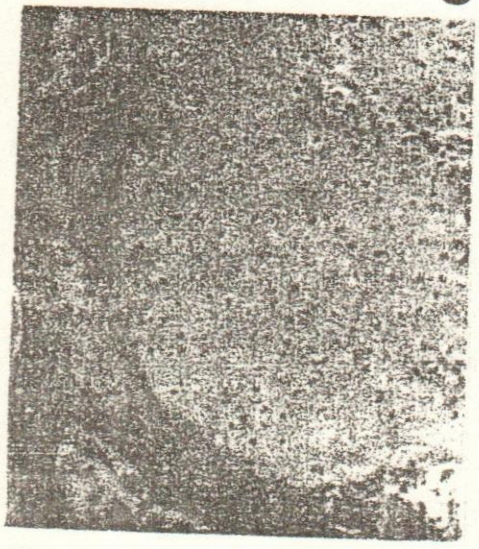
Fig. (4): Showing cystic formations and necrosis of most layers of epidermis of lips. H&E. Stain X 63.

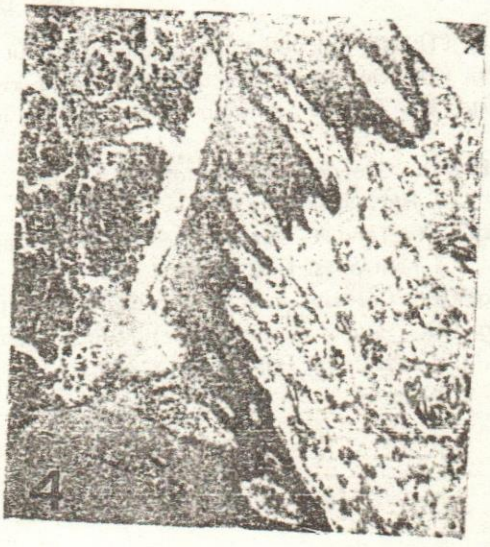
Fig. (5): Tongue showing ulcerative lesions infiltrated by mononuclear cells and few neutrphils. H&E. Stain X 160.

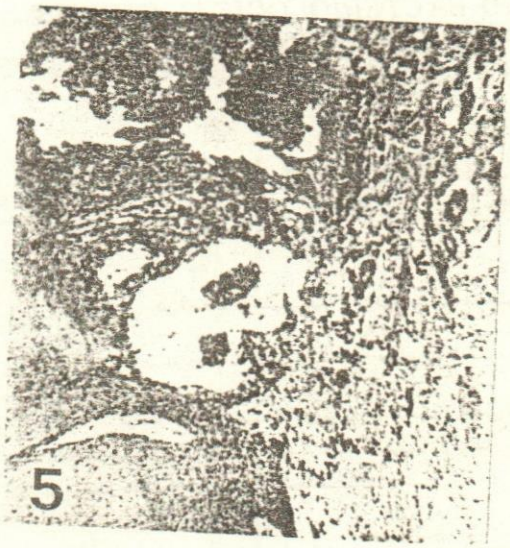
Fig. (6): Cardiac muscle showing degeneration and haemorrhages. H&E. Stain X 400.

Fig. (7): Heart muscle showing focal spots of small round cells infiltration and few eosinophils H&E. Stain X 400.

FOOT MOUTH DISEASE, BUFFALOES







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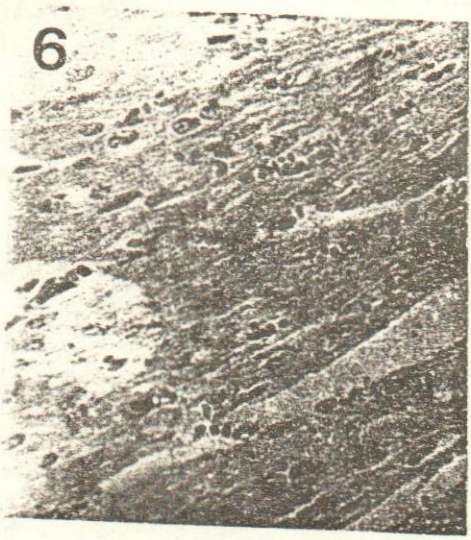

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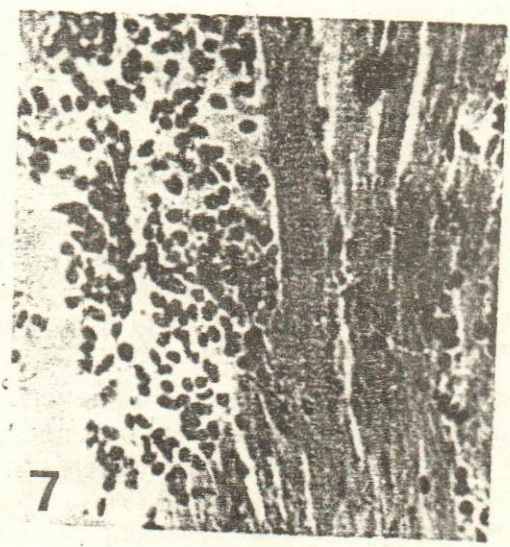

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CLINICO-PATHOLOGICAL AND BACTERIOLOGICAL STUDIES ON AVIAN PASTEURELLOSIS

(With 6 Figures)

By

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دراسات إكلينيكية ، باثولوجيـة وبكتريولوجية عن كوليرا الطيـــور

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لوحظ حدوث عـدة أوبئـة متكـررة لمـرض كوليـرا الطيـور فـي مزارع الأمهـات . كما تم عزل وتصنيف الميكروب المسبب وكذلك وصفت صورة المرض إكلينيكيا وباثولوجيـا وصفا دقيقا نوقشت النتائـج .

SUMMARY

Several outbreaks of fowl cholera were recorded as endemic recurrent epornitics in breeder poultry farms. The causative organism was isolated and serologically identified as 5 and 8 : A serotypes of Pasteurella multocida. Clinically, an acute intermittent from as well as a long lasting chronic one were observed. Postmortem and histopathological findings were described and discussed.

INTRODUCTION

Fowl cholera (avian pasteurellosis) or avian haemorrhagic septicaemia is a highly contagious disease affecting many species of domesticated and wild birds. The disease occurs enzootically in most of countries resulting in higher losses (CARTER and BAIN, 1960). Several serotypes of Pasteurella multocida (A-O) have been serologically identified by plate agglutination or passive immunization tests (HEDDLESTON, 1962).

Usually, the disease runs an acute course with high morbidity and mortality rates, or followed by a chronic one (RHODES and RIMLER, 1984). Signs of acute infection are only observed few hours before death, while the chronic form may follow the acute phase resulting in localization of the infection (SIRBU et al., 1971). In the acute form of the disease, septicaemic lesions are constantly observed on necropsy, while in the chronic one, the lesions are mainly localized in wattles and respiratory passages,

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