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تأثير المهبط للمناعة بعد الاصابة بمرض الاسهال الفيروسي في العجول الجاموسي بصعيد مصر

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درست الاعراض الاكلينيكية وصورة خلايا الدم والصورة التشريحية المرضية ومناعـة الجسم لحالات مرضية مصحوبة بأعراض تنفسية وهضمية لوباء في مزرعة للعجول الجاموسي خلال عام ١٩٨٤، ١٩٨٥ بالقرب من مدينة المنيـا• وقد أظهرت النتائج انخفاضا ملحوظا في العد الكلي لكرات الدم البيضاء في مراحل المرض الأولى سرعان ماتحول الى زيـادة ملحوظة في هذا العدد مع انخفاض العد الكلي لكرات الدم الحمراء وتركيز هيموجلوبـين الدم في معظم الحالات ، وبالتشريح المرضي كانت الصورة مشابهة للاصابة بمـرض الاسهال الفيروسي للماشية ، وقد نوقشت الحالة المناعية للحيوانات المصابة•

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IMMUNOSUPPRESSIVE EFFECT OF BOVINE VIRAL DIARRHOEA MUCOSAL DISEASE (BVD-MD) IN BUFFALO CALVES IN UPPER EGYPT

(With 4 Tables and 4 Figures)

By
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SUMMARY

Clinical, haematological, P.M. and virological studies were conducted on buffalo calves farm (430) aged from 2-4 months, near Minia City, Upper Egypt during the prevalence of an outbreak (December 1984- Feberuary, 1985) accompanied by respiratory and alimentary manifestations. Leucopenia was reported at the earily stage of illness, however leucocytosis was observed later on associated with lowered values of total red cells count and Hb concentration in most of diseased cases. Post-mortem examination revealed the presence of BVD-MD virus picture. No antibodies were detected by agar-gell ppt. test.

INTRODUCTION

The bovine viral mucosal disease complex constitutes a costly and troublesome disease problem in bovine industry. The extent of financial loss due to bovine mucosal disease complex remains largely unmeasured, but it is generally agreed that annual losses are quite heavy and the disease has world wide prevalence (RAMSEY and CHIVERS, 1953). The mucosal disease complex includes bovine viral diarrhoea, rinderpest, blue tongue, papular stomatitis, malignant catarrhal fever and miscellaneous causes of bovine stomatitis (KAHRS et al., 1971). Bovine viral diarrhoea (BVD) is a disease caused by the bovine viral diarrhoea virus (BVDv) which is easily transmitted. It was first recorded in the United States (OLAFSON et al., 1946). Concurently similar cases with variations in degree of severity, chronicity and sporadicity were described and named mucosal disease which is usually (but not always) accumpanied with severe diarrhoea, persistent excessive lacrimation, ulceration or erosions of the oral mucosa (RAMSEY and CHIVERS, 1953 and RAMESEY, 1956).

The BVDV has an affinity for lymphocytes and rapidly dividing cells. Thus it causes leucopenia and lymphoid depletion in lymph nodes and Peyer's patches. It has been suggested that BVDV infection has an immunosuppressive or immunodepleting effect (JOHNSON and MUCOPLAT, 1973).

HOPKINSON et al. (1979) demonstrated that antibody response to infection with BVDV can be detected by gell diffusion test. HAFEZ (1973) isolated and identified bovine viral diarrhoea mucosal disease virus in Egypt. NAFIE et al. (1984) indicated the presence of BVD/MD virus among fattening calves at Assiut Governorate. Therefore the aim of this work

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was to study the role played by BVD/MD virus infection in the occurance of present diseased condition, the extent of pathological and blood picture changes as well as the serological detection of antibodies against BVD/MD virus.

MATERIAL and METHODS

The study was carried out on 430 buffaloe calves aged 2-4 months old at Towa Village near Minia City, Upper Egypt. These buffaloe calves were purchased at Minia Governorate markets from variety sources. Some indviduals were seen to have inappetence, salivation, hyperaemia of conjunctival mucosa and oculo-nasal serous discharges. The rest of the group was carefully examined. Calves with apparent abnormal clinical signs or with elevated body temperature were soon isolated. Both strict hygeinic measure as well as therapeutic trials for medical treatment were applied.

Anticoagulated blood samples representing various stages of the disease were collected on EDTA. These samples were used for haematological picture. Red blood cells (T/L), Hb (gm/l) and total W.B.Cs (G/L) were determined using Electronic Cell Counter (Cell Dyne 300 Sequoia Turnor). P.C.V. and differential leucocytic count were estimated according to the routine methods of haematology. Another blood samples were taken for the separation of serum and used fer serological examination. Serological agar gell ppt. test was carried according to HOPKINSON et al. (1979). Dead and sacrificed severly affected animals were carefully examined. The alterations were recorded and photographed.

RESULTS

The obtained results for clinical and haematological examinations are illustrated in tables 1, 2, 3 & 4. In table (1) daily mortalities rate among diseased calves is presented while in table (2) apparent abnormal clinical signs (elevated body temperature, mouth lesions, lacrymal and nasal discharge and diarrhoea) were presented in various stages of the diseased condition.

From table (3) it appears that haemoconcentration was evident in groups I & II while haemodilution was characteristic for group III. Red blood cells indices (MCV, MCH & MCHC) were consequently variable between diseased groups (Table 3).

Marked leucopenia was generally evident among all groups however it was more obvious in group I & II. Lymphopenia was a conistant findings with neutrophilia (Table 4).

Unsegmented neutrophilia was characteristic in but all diseased groups. Variations in easinophils, basophils and monocytes count were inconsistent.

All the tested serum samples were negative to BVDV (no ppt. line was formed between virus and the sera) as well as to rinderpest virus as performed by S.T. test.

Post-mortem examination of dead calves revealed that they were severely dehydrated with the evidence of profuse diarrhoea. Multiple erosions, usually 1-5 cm in diameter were recorded in the mucosa of the muzzle, buccal cavity, tongue, oesophagus, abomasum and small intestines. Oral lesions were seen on the inner surface of lower lip, at the commissures of the mouth. Some appear on tip of the tongue. In few cases the lesions were so severe that it rendered teeth so loose and ended in its destruction. Congestion and hyperaemia was evident allover the internal organs (heart, lung, liver, spleen and kidneys). Some indvidual have had purrulent pneummia with adhesion to pleura. Oesophagal erosions were characterastically arranged in linear arrays. Gall bladder was markedly enlarged and filed with dark viscid bile.

DISCUSSION

Severe to fatal bovine virus diarrhoea was evident among tested herd. The age of the animals played a determinal factor in infection. Within a period of 10.12.1984 to 1.2.1985, 183 calves were dead from a total of 430 indviduals with a ratio of 42.56% mortality rate. Time of occurance (Winter months), where environmental temperature could reach 20°C at morning and sometimes zero at night, evidently aggreviated the severity of the disease. It was emphasized by KAHRS et al. (1971) that BVD developped during all seasons. RAMSEY and CHIVERS (1953) identified mucosal disease in feedlot cattle with low morbidity and high mortality. OLAFSON et al. (1946) reported that a larger percentage of animals in herd were involved and cattle of all ages were affected. It was suggested by MALQUIST (1968) that the prepondance of clinical signs among young cattle between 4-24 months age may reflect the ubiquity of infection and its modulation by the presence of colostrum-conferred antibody or an actual age related susceptibitlity (KAHRS et al., 1971).

Similar outbreaks were recorded by GREIG et al. (1981) in England among 4-9 months old crossbred calves. Higher mortality rate (20-40%) was recorded by BAZ et al. (1982) while a rate 100% mortalities was registered at Kena Governorate by the same authors and by EL-SEBAIE et al. (1985) at Assiut where mortality rate amounted 30% and morbidity rate 70%.

The clinical signs in the present study were generally resembling those previously reported by BAZ et al. (1982) EL-SEBAIE et al. (1985). KAHRS et al. (1971) indicated that, however when totally susceptible populations are infected, the mortality and morbidity rates can be impressive. Thus a variety of clinical signs patterns, varied in severity from inapparent non clinical infection or mild febrile disease to an acute fatal syndrome could appear. Chronic debilitating infection can also occurs. the severity and outcome of the disease may be dependant upon the degree of activation of the immune system.

The BVDV probably entered new hosts through the alimentary and respratory systems. Virions infect epithelial cells in nose, mouth, abomasum and intestines and therein replicate. Viraemia persists during febrile stages and usually terminates when antibodies reach significant levels.

Haemoconcentration was evident in groups I & II (table 3) due to the dehydration observed in both groups. Red blood cells in these groups are microcytic and hyperchromic in nature. For the third group, lowered total red blood cells count with respective dropped P.C.V. and Hb concentration was evident. Anaemia here is macrocytic hyperchromic. Similar observations were recorded in respective diseased conditions (INABA et al., 1970 and EL-SEBAIE et al., 1984).

Regarding white blood cells picture, it appeared that leucopenia was evident in groups I & II. Such condition emphasizes that the primary cause of illness was a viral agent which is usually accompanied by leucopenia (SCHALM, 1979 and COLES, 1980). Similarily KAHRS et al. (1971) stated that profound leucopenia is usually present particularily in the earily stage of the infection. Total white blood cells count was observed to return to normal levels 14 days post-experimental infection (SCOTT et al., 1973). The third group had rather normal total white blood cells count, suggesting secondary bacterial invadors (SCHALM, 1979; COLES, 1980 and EL-SEBAIE et al., 1984).

Lymphopenia accompanied by neutrophilia and increased unsegmented cells count was a characterstic finding in all diseased groups however it was well obvious in the 1st & IInd groups. BVDV enters lymphoid tissue either through lymphatic or blood vessels. In nodes,

spleen and Peyer's patches, cells are destroyed and lymphocytes become depleted. Similar results were previously reported by ROTH et al. (1981) who observed marked neutropenia with eosinopenia following experimental BVDV infection.

The presence of epithelial defects, seen at necropsy, in the buccal cavity simulates the finddings previously reported by NAFIE et al. (1984) and EL-SEBAIE et al. (1985). In the present study the bacterially complicated erosions showed suppurative inflammatory reactions and subsequently the erosions were so deep that the teeth were loosened.

From the results of serological tests, it is concluded that all tested sera samples were BVDV negative. S.T. test for rinderpest virus was also negative. Failure of individual susceptible cattle to produce antibody when infected may result from immune tolerance, immune paralysis or immune suppression (CORIA and McCLURKIN, 1978). The hypothesis of specific immune tolerance (failure of calf to recognize BVDV as foreign because prenatal infection occured during the development of the recognition phase of its immune system) has been diffecult to substantiate because efforts to produce the syndrome experimentally have resulted in abortion or prenatal development of actively induced humoral antibody (GRATZEK, 1968). Immunosuppression has been demonstrated in association with calves persistently infected with BVDV. The question remains if immunosuppression is an enabling factor in the persistent BVDV infection or does the persistent infection cause the immunosuppression (JOHNSON and MUCOPLAT, 1973).

From the abovementioned results, we can conclude that further research on BVD/MD virus is needed to elaborate the immunosuppressive effect (if any) of field and vaccine BVDV strains and immunologic responses of buffaloe to the virus. The possible effect of BVDV induced immunosuppression shares in lowering resistance of bufaloe to other infection requires also further study.

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Table (1)

Daily Mortalities among diseased calves

Date	No. of mortalities	Date	No. of mortalities	Date	No. of mortalities	Date	No. of mortalities
10.12.84	2	24.12.84	5	7.1.85	5	21.1.85	2
11.12	2	25.12	3	8.1	5	22.1	5
12.12	-	26.12	5	9.1	5	23.1	-
13.12	8	27.12	8	10.1	4	24.1	2
14.12	7	28.12	6	11.1	-	25.1	-
15.12	3	29.12	2	12.1	3	26.1	1
16.12	4	30.12	7	13.1	-	27.1	-
17.12	10	31.12	4	14.1	6	28.1	2
18.12	-	1.1.1985	3	15.1	1	29.1	-
19.12	6	2.1	2	16.1	3	30.1	1
20.12	8	3.1	5	17.1	3	31.1	2
21.12	3	4.1	-	18.1	5	1.2.1985	5
22.12	3	5.1	4	19.1	4		
23.12	8	6.1	1	20.1	-		

Table (2)

Basic Clinical Manifestations

	No. of selected animals	Body temp.	Mouth lesions	Lacrymal discharge	Nasal discharge	Diarrhoea
Group I (Acute)	10	41.00±0.15	+++	++	++	+
Group II (Subclinical)	10	40.00±0.53	+++	++	++	-
Group III (Convalesent)	10	39.20±0.33	++	++	++	-
Group IV Clinically healthy	10	38.84±0.20	-	-	-	-

Table (3)

Red Blood Cell Picture

	T.R.Bcs (T/L)	P.C.V. %	Hb (Gm/L)	M.C.V. (Fl.)	M.C.H.	M.C.H.C. (Gm/dL)
Group I	12.87±1.34	42.25±4.02	291.90±12.35	32.85±1.41	22.8±0.67	70.1±1.06
Group II	13.02±3.13	41.71±5.72	267.87±13.11	33.63±3.75	21.56±2.28	64.46±3.09
Group III	9.35±1.47	39.80±1.71	146.75±9.23	44.75±4.70	15.88±2.01	38.66±2.87
Group IV	11.13±1.24	40.00±2.63	177.30±5.43	36.18±5.35	16.20±2.72	44.54±4.03

Table (4): Total and differential leucocytic count

	T.L.C. (G/L)	Differential Leucocytic Count					
		Lymph. %	Band %	Segmented %	E sin. %	Baso. %	Mono. %
Group I	7.95±0.89	48.00±2.48	13.50±1.89	34.75±2.38	3.25±1.02	0.50±0.20	-
Group II	7.77±0.68	45.00±5.18	7.71±1.09	45.43±4.15	1.00±0.33	-	-
Group III	9.00±0.81	56.60±4.87	6.80±1.43	36.60±3.60	-	0.30±0.15	0.50±0.32
Group IV	9.80±0.78	69.40±8.56	1.40±0.4	28.40±3.91	0.60±0.30	-	0.20±0.10

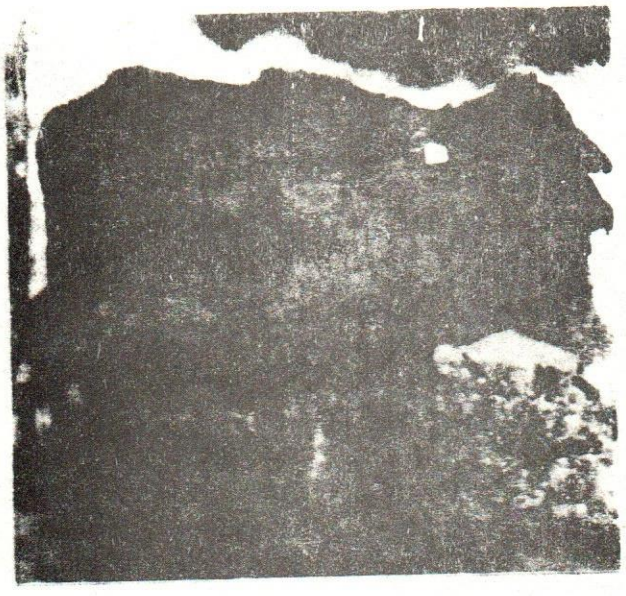

Fig. (1): Signs of dehydration

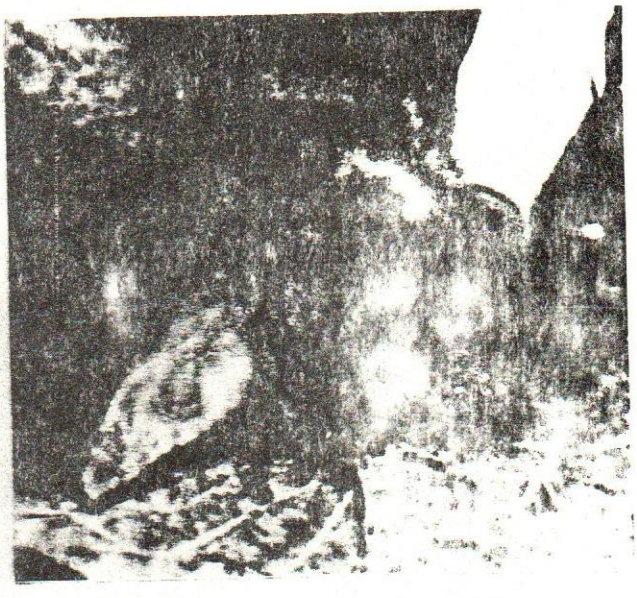

Fig. (2): Lacrynal discharge

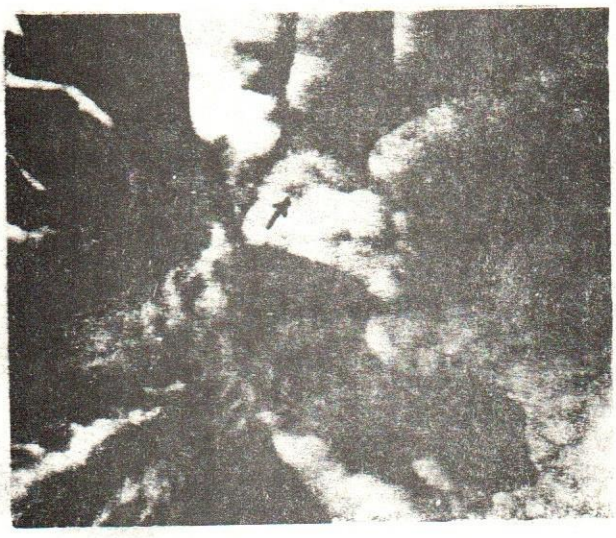

Fig. (3): Minute oral lesion on the inner side of upper lip.

Fig. (4): Severe mouth lesions in the inner side of upper lip.