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**SOME INVESTIGATIONS ON AN OUTBREAK OF ENZOOTIC
BRONCHOPNEUMONIA AMONG FATTENING BUFFALO-CALVES**
(With 2 Tables & 4 Figs.)

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بعض الفحوص على وباء التهاب الشعب والرئة المتوطن بين
عجول التسمين الجاموس

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انتشر وباء التهاب الشعب والرئة بسبب اعياء ونفوق عليتين فيما بين
عجول التسمين الجاموس في مزرعة كلية الطب البيطري جامعة قناة السويس . اشتملت
أهم الأعراض الاكلينيكية على صعوبة التنفس وافرارات أنفية مخاطية وكحة وأصوات
تنفسية غير عادية مع ارتفاع درجة حرارة الجسم . أظهر الفحص الباثولوجي أن هناك
أعراض عديدة من الالتهاب تفاوتت بين الحالة المزمنة والمتكررة كما لوحظ وجود
خراجات منتقلة في الرئة . أظهر العزل الميكروبي اصابة هذه العجول بميكروب
الكواريني الصديدي والعضوي المعوي والميكوبلازما . كما أوضحت التحاليل الدموية
اصابة الحيوانات بالأنيميا وارتفاع عدد الخلايا البيضاء في الدم في بعض الحالات
بينما نقصت في حالات أخرى . كما أوضحت التحاليل الكيميائية ارتفاع قيمة الجلوكوز
والجلوبيولين في الحيوانات المصابة . اتخذت الاجراءات العلاجية والصحية اللازمة
وبها توقف المرض من الانتشار بين الحيوانات الغير مصابة بينما الحالات التي أصيبت
لم تستجب للعلاج .

SUMMARY

An outbreak of enzootic bronchopneumonia with high morbidity and mortality rates was recorded in fattening buffalo calves. The major clinical manifestations were dyspnoea, mucopurulent nasal discharges, coughing and abnormal respiratory sounds with elevated body temperature. Chronic necrotizing suppurative pneumonia, gangrenous pneumonia as well as metastatic abscesses were recorded in pathological examination of pulmonary tissue. Such changes were closely related to the

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period of illness. Subacute and/or chronic interstitial nephritis was evidenced pathologically in some affected animals. Corynebacterium pyogenes, Haemolytic E.coli and Mycoplasma Sp. were isolated from nasal swabs & pulmonary tissues. Microcytic hypochromic anaemia was revealed in diseased animals. Leucocytosis was evidenced in some affected animals while leucopenia was noticed in some other affected calves. Hyperglycaemia and hyperglobulinaemia were noticed in diseased animals. Preventive and therapeutic measures stopped the progression of the disease among the clinically healthy heardmates, while in advanced cases recovery was difficult and all chronic advanced cases were completely destructed.

INTRODUCTION

Respiratory affections constitute one of the most important factors causing serious losses among buffalo-calves. Most of these affections are characterized by high morbidity and mortality especially in intensive rearing systems. The adverse effect of respiratory diseases on calves growth rates and utilization of food was observed by SCHMOLDT et al. (1979) who reported that the weight gain was decreased in calves with bronchopneumonia in comparison with the control healthy ones.

Several environmental factors including hunger, system of weaning, extremely hot or cold climatic temperature and bad hygienic conditions in housing, might act as stress factors lowering the resistance of calves and making them liable to several infectious agents and predispose for outbreaks of respiratory affections (JENSEN & MACKEY, 1979; BLOOD, et al. 1986).

Parainfluenza-3, adenovirus, I.B.R., virus R.S.V. were demonstrated in bovine respiratory diseases (WISEMAN, et al. 1980; PIRIE, et al. 1981; EL-SEBAIE, et al. 1984; VERHOEFF, et al. 1984 and NAFIE, et al. 1984). Pasteurella multocida, P. haemolytica and Corynebacterium pyogenes, were the most common bacterial pathogens associated with bronchopneumonia in cattle (WIESMAN, et al. 1976; BRYSON, et al. 1979; AL-ALLAWY, et al. 1979). Moreover, Staph. aureus; Strept. pyogenes; E.Coli and Corynebacterium pyogenes were isolated from buffalo-calves suffering from respiratory manifestations, (ELYAS, 1982).

Giant cell pneumonia was described in an outbreak of respiratory affection in buffalo calves (MOUSTAFA, et al. 1975), Exudative interstitial pneumonia was observed in 6-10 months old buffaloe calves suffering from respiratory affections (AL-ALLAWY, et al. 1979). Furthermore, HUSSEIN (1979) recorded that the percentage of fibrinous,

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proliferative, interstitial, bronchopneumonia and granulomatous pneumonia in calves, were 13, 17, 14, 10 and 20%, respectively.

In the animal farm station of the Faculty of Vet. Med., Suez Canal Univ., it was observed that fattening buffalo-calves were admitted to the clinic in increasing number. Such animals showed signs of respiratory affections. A considerable number of them died or emergency slaughtered.

This work is aimed to investigate this problem from the clinical, bacteriological and pathological points of view, as well as to investigate some haematological and biochemical changes in such cases.

MATERIAL and METHODS

Animals:

450 fattening buffalo calves at the animal farm station of the Faculty of Vet. Medicine, Suez Canal Univ. of 250-350 Kg Bw, constitute the material of this work. Animals were purchased from different localities in Delta and around the province at January 1991 for the purpose of veal production. On arrival routine clinical examination was performed on all calves including clinically affected calves and twenty clinically healthy herdmates which used as control.

Samples:

Anticoagulated blood samples were collected for determination of total erythrocytic count (TEC), total leucocytic count (TLC); Haemoglobin (Hb) and Haematocrit (Ht). Serum samples were obtained for biochemical analysis.

Specimens of lungs and kidneys were taken from emergency slaughtered and dead animals for pathological examination. The gross findings were recorded.

Nasal swabs and parts of the lungs of emergency slaughtered animals were aseptically collected for bacteriological examination.

Methods adopted:

Calves were examined clinically, body temperature, respiratory and pluse rates were recorded. Respiratory system was fully examined, Rumen and heart were also examined (KELLY, 1984).

T.E.C. & T.L.C. were performed using improved haemocytometer. Haematocrit was determined using microhaematocrite method (JAIN, 1986). Haemoglobin concentration, serum total protein, albumin and serum glucose were determined colorimetrically

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using test kit after VANKAMPEN (1961); WEICHSELBAUM (1946); DRUPT (1974) and TRINDER (1969) respectively. Serum globulin level calculated mathematically and A/G ratio were calculated (COLES, 1986).

Lung & Kidneys tissue specimens of dead and emergency slaughtered animals, were fixed in 10% neutral buffered formaline and dehydrated by ascending grades of alcohols, cleared by using xylol. Paraffin sections were made in a thickness of 4-6 μ (DRURY and WALLINGTON, 1980) and stained with Harris haematoxyline and eosin (HARRIS, 1898). Gross and microscopic changes were recorded and photographed.

Statistical analysis was carried out using T-test according to KALTON (1967).

A control program was outlined including isolation of the affected animals in a clean, well ventilated yards. The clinically healthy calves were turned to another clean, disinfected yard and given (i.m) two therapeutic doses of long acting terramycin*, with an interval of 72 hrs. The same program was applied on the diseased animals. The dirt and soil of the infected yards were removed to a depth of 120 cm, then a layer of limestone was evenly distributed and then covered by dry layer of sandy soil. Water troughs were completely drained, cleaned and washed by solution of potassium permanganate 1:1000.

RESULTS

Clinical findings:

Anorexia, dullness, pale mucous membranes and weakness were noticed on the majority of the affected animals. Pyrexia (40-41.5°C) was evidenced at the onset of signs and lasted for 2-3 days, while the majority of the affected calves showed normal or subnormal body temperature thereafter. Bilateral nasal discharges were constant findings in all diseased calves which was slight to copious in quantity, serous to muco-purulent in nature. Moreover, bilateral frothy purulent nasal discharges were observed in advanced cases. Coughing was also a prominent sign at the beginning of the outbreak which was harsh, frequent and as the disease becomes worse, it became infrequent and soft. Dyspnoea with respiratory snoring, mouth breathing and grunting were frequently observed just before death or emergency slaughter. In other less severely affected calves, an increase in the rate and depth of respiration was noticed. Hypervesicular sound and dry rales were detected at the beginning of the outbreak. Different abnormal respiratory sounds such as moist rates, dry rales and

* Contain 200 mg oxytetracycline base per ml (Pfizer).

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crackling sounds were audible on auscultation of the lungs of diseased animals in progressed cases. Arthritis with lameness was evidenced in some affected calves.

Pathological findings:

Lungs:

a) Macroscopic picture

Both sides of the lungs were affected, the most anterioventral parts of the apical lobes and in some cases the cardiac and diaphragmatic lobes were consolidated with a patchy distributed and surrounded by areas of compensatory emphysema.

The colour of the pulmonary tissue ranged from red, dark red, grey and bluish. Some affected lungs showed greenish black discolouration. The greenish black parts emerge offensive smell. Multiple variable-sized abscesses were observed all over the pulmonary surfaces. The cut surfaces of the affected parts of the lung were bluish red in colour and showed multiple abscesses varying in size. Its distribution regularly accompanied the bronchial tree in some cases, while in other cases it was distributed along with the bronchial tree in addition to some haphazardly distribution from which yellowish-creamy pus discharges.

b) Microscopical picture:

The pulmonary tissue revealed a wide variety of pneumonia. Chronic necrotizing suppurative bronchopneumonia (Fig. 1), fibrinous and serofibrinous pneumonia, metastatic abscesses as well as gangrenous pneumonia were all recorded.

In chronic necrotizing suppurative bronchopneumonia, the bronchi and bronchioles showed advanced necrosis of the epithelial lining which was completely replaced by liquified tissue. The luminae contained large amounts of pus which is formed from necrotic tissues, neutrophils, pus cells, tissue debris and bacterial colonies (Fig. 2). The lamina propria was replaced by basophilic substances contained pus cells, neutrophils, plasma cells, macrophages and lymphocytes. The adjacent alveoli showed pulmonary oedema and inflammatory cells.

Some areas showed multiple abscesses formation in the pulmonary parenchyma faraway from bronchi or bronchioles.

The interlobular septa and some parts of the pulmonary tissues showed fibrinous and serofibrinous exudate. The involved parenchyma revealed the presence of eosinophilic granular substances mixed with eosinophilic threads or network.

Some affected lungs revealed gangrenous pneumonia characterized by presence of faint eosinophilic structureless mass, gas vacuoles, haemosidrosis and long bacilli (Fig. 3).

Kidneys:**a) Macroscopical picture:**

The kidneys of the affected animals showed no significant macroscopical changes except in certain individuals, where the kidneys showed a whitish areas scattered all over the kidney particularly in the cortico-medullary junction.

b) Microscopical picture:

The kidney tissue of some affected animals showed subacute and/or chronic interstitial nephritis. The involved tissue revealed leucocytic infiltration by the mononuclear cells as lymphocytes, macrophages, plasma cells and some cases contained fibroblasts and fibrous connective tissue proliferation of interstitial tissues. The adjacent renal tubules revealed atrophy of the collecting and some renal tubules (Fig. 4).

Bacteriological Examination:

Corynebacterium pyogenes, Haemolytic E.coli and Mycoplasma sp. have been isolated from nasal swabs and pulmonary abscesses.

Haematological picture and serum biochemical analysis were shown in tables 1 & 2 respectively.

Control Measures:

The strict preventive and therapeutic measures stopped the progression of the disease among the clinical healthy individuals completely and no further cases were detected. However, in diseased animals the therapeutic effect was dependant on the progression of lung lesions. In early acute cases the treatment was highly effective but in chronic conditions the recovery was fairly achieved where the response to treatment was very poor.

DISCUSSION

The development of respiratory diseases in calves in the farm under investigation was predisposed by several factors including lack of proper hygienic measures inside the animal yards. Mud and dirt were accumulated under shelters and around water troughs and even on calves which make the environmental and managerial practices suboptimal. Lack of adequate feed additives or green fodder is an additional factor make the calves more susceptible to infection.

The investigations revealed a morbidity rates amounted 36% and a mortality rates 5%. About 10% of the affected animals were emergency slaughtered.

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The significant general signs observed in this outbreak were reduced appetite and dullness, variable body temp. as some animals were pyrexia in early stages while normal to subnormal body temperature was recorded in advanced cases. Cough, mucoid to mucopurulent, bilateral nasal discharges, mouth breathing with moderate to severe dyspnoea were evidenced. Furthermore, abnormal breath sounds were detected by auscultation of the affected lungs. The clinical observations were coincided with that previously recorded by AL-ALLAWY, et al. 1979; EL-SEBAIE, et al. 1984 & 1987 in similar outbreak of respiratory affection in fattening buffalo-calves in Upper Egypt.

The bacteriological and pathological findings showed the complex nature of the outbreak as Corynebacterium pyogenes, Haemolytic E.coli and Mycoplasma sp. were isolated from the pulmonary tissues of emergency slaughtered animals as well as from the nasal swabs of the affected ones. Furthermore, the pathological examination revealed, chronic necrotizing suppurative bronchopneumonia accompanied with fibrinous and serofibrinous pneumonia, metastatic abscesses as well as gangrenous pneumonia. These findings explain the progressive non curable nature of the individual that not responded to treatment in addition to the severe dyspnoea and the enlarged area of non functioning pulmonary tissues. Similar bacteriological and pathological findings were reported in fattening buffalo-calves suffering from respiratory affection by RAMSEY, et al. (1966); MOUSTAFA, et al. (1975); EL-SULTAN, et al. (1987) and AL-ALLAWY, et al. (1979).

The isolated Mycoplasma sp. and Corynebacterium pyogenes may explain the chronic suppurative non curable nature of the disease as postulated by BLOOD, et al. (1986) and STALHEIM (1983) who stated that the clinical signs usually appear only when pulmonary involvement reaches a sufficiently advanced stage. The authors added that, environmental stresses and/or infection by other bacteria or viruses are necessary for establishment of infection with Mycoplasma and the development of clinical signs.

Regarding the haematological picture, a significant ($P/0.05$) decrease of total red cell count, Hb. concentration and haematocrit, were recorded in affected animals in comparison to the clinically healthy herdmates. Leucocytosis was observed in diseased buffalo calves as shown in Table 1. Red cell indices revealed a microcytic hypochromic anaemia which may be secondary to inflammatory diseases, and might be attributed to sequestration of iron in the bone marrow macrophages and hepatocytes during the infection, thus become unavailable for utilization in haemoglobin synthesis, resulting in inhibition of erythropoiesis (BEISEL, 1976; COLES, 1986; JAIN, 1986). The recorded leucocytosis might be attributed to stimulation of bone marrow stem cells by bacterial mediators increasing neutrophil production (COLES, 1986 and JAIN, 1986).

Similar haematological picture was recorded by AL-ALLAWY, (1979); AMER, et al. (1979) and EL-SAYED (1990) in fattening buffalo-calves suffering from respiratory affections.

Serum biochemical analysis revealed highly significant increase of total protein and globulin. This may be attributed to chronic antigenic stimulation and hypergammaglobulinemia (WHITLOCK and WINGFIELD, 1980). Significant hyperglycemia recorded in diseased animals may be due to the anoxia that associated the chronic bronchopneumonia as postulated by ANDERSON (1980) who stated that hyperglycemic response is characteristic of ruminants with severe life-threatening diseases. Furthermore COLES (1986) reported that hyperglycemia may result under anoxic conditions, since liver glycogen is relatively unstable in the presence of a deficient oxygen supply.

It can be concluded that several factors were incriminated in respiratory affections such as the predisposing factors which include, bad hygienic conditions, deficiency of micronutrients and the crowding which inversely affect on the immune balance of the herd. The primary control of these factors will protect the herd from similar affections. This was clearly defined after the application of the control program in the farm. The therapeutic effect of any drug depends on the progression of the lung lesion from one hand and on the response of the secondary invaders to antibiotics from the other hand. In cases of Mycoplasma infection there was no response for treatment.

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Table (1) : Haematological picture in clinically healthy and diseased buffalo-calves.

Item	Unit	Control (20)			Diseased (65)		
		Min.	Max.	Mean±S.E.	Min.	Max.	Mean±S.E.
Hb.	gm%	11.8	14.9	12.3±0.85	5.5	10.2	8.6±0.54 **
P.C.V	%	31.0	38	36.5±0.8	18.0	28.0	22.3±0.6 **
T.R.B.Sc.	X 10 ⁶ /μl	6.1	9.4	8.2±0.2	5.2	8.0	6.6±0.38 **
M.C.V.	Pg	35.0	42.6	35.7±1.2	26.0	35.2	28.3±1.5 *
M.C.H.	Fl.	11.5	18.2	13.5±0.8	10.0	17.8	14.7±0.7 N.S
M.C.H.C.	%	28.3	36.0	34.2±0.9	24.3	35.2	32.5±1.4 **
T.L.C.	X10 ⁹ /μl	5.9	11.6	8.9±0.7	9.8	14.6	13.2±0.4 **

** highly significant at p < 0.01

* significant at p < 0.05

N.S. Non significant

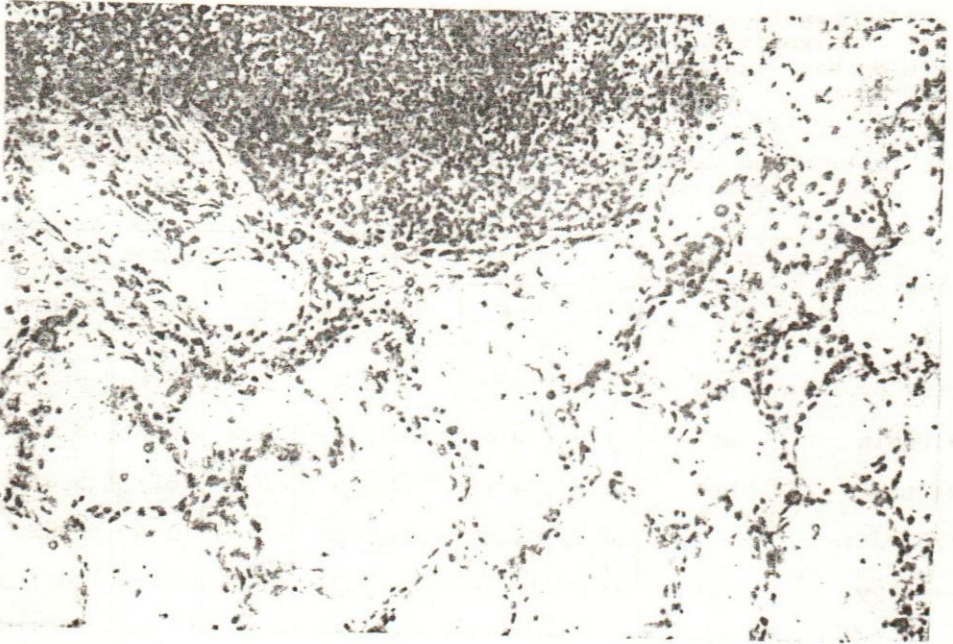
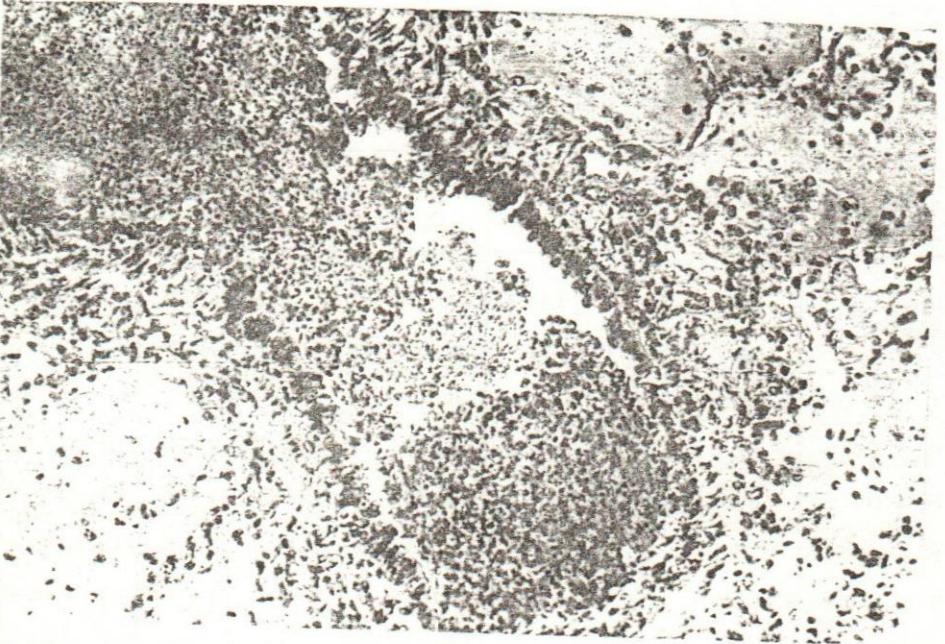
Table (2) : Serum biochemical analysis of clinically healthy and diseased buffalo-calves.

Item	Unit	Control (20)			Diseased (65)		
		Min.	Max.	Mean±S.E.	Min.	Max.	Mean±S.E.
Total protein	gm%	5.0	7.9	7.1±0.4	5.4	13.6	9.2±0.3 ** N.S
Albumin	gm%	1.8	3.6	3.4±0.4	2.2	4.8	3.8±0.13 **
Globulin	gm%	2.2	4.1	3.7±0.4	2.0	6.7	5.3±0.22 N.S
A/G ratio	--	0.52	1.27	0.95±0.05	0.41	2.0	0.98±0.09 *
Glucose	mg%	65.8	110.4	75.0±3.5	58.3	167.8	140.1±3.2 *

** highly significant at p < 0.01

* significant at p < 0.05

N.S. Non significant



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