PASTEURELLOSIS IN OSTRICH

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Abstract

This study was carried out on two ostrich farms suffering from high morbidity and increased mortality rates with signs of respiratory distress, difficult breathing, purulent nasal discharge, cough, anorexia, neck dropping, and in addition to death. Complete postmortem examination, bacteriological and histopathological examinations were done, as well as antibiotic sensitivity test of the isolated bacteria and treatment regimens were applied.

The result of postmortem examination revealed pulmonary congestion, consolidation and brownish patches on the lung; the trachea was occluded with bloody exudates. *Pasteurella multocida* was recovered by (90%) from most examined cases, identified biochemically, and by using pathogenicity test . *Klebsiella pneumonia, E.coli* and *Edwardsiella* spp. were also isolated with variable percentages. Histopathological examination revealed severe pathological changes in the lungs characterized by pleuritis serofibrinous pneumonia and various degenerative changes in other internal organs . Antibiotic sensitivity test showed that all *Pasteurella multocida* isolates were highly sensitive to Trimethoprim (TMP)/ Sulphamethoxazol (SMx), Ceftiofur and Amoxicillin (100%), and less sensitive to Tetracycline (93%) and Enrofloxacin (80%). Different treatment regimens were applied based on sensitivity results .A mixture of (TMP) & (SMX) at a concentration of 8 mg/ml and 40 mg/ml, respectively in drinking water for 5 successive days was found to be more effective in improving clinical signs and control of mortality rate.

INTRODUCTION

Pasteurellosis or fowl cholera naturally infects over 100 species of wild birds and typically occurs in explosive outbreaks, which kill hundreds to thousands of birds (Botzler, 1991). Pasteurellosis refers to pneumonia, septicemia and other infection

caused by bacteria of genus Pasteurella. Pasteurella spp. especially *P.multocida* and *P.hemolytica* have been incriminated as important pathogens of both domestic and wild birds (Rimler and Glisson, 1997), resulting in severe economical losses among poultry population (Dunbar *et al.* 2000) .The occurrence of Pasteurellosis in ostrich is rare (Okoh, 1980 and El-faki *et al.* 2002). In Egypt, Rabab (2003) isolated *Pasteurella hemolytica* from ostrich farms at age ranging from 1 week to 3 months. Outbreaks of pasteurellosis are often associated with climatic stress and possible predation in free range. The severity of infection is usually controlled by strict sanitation and biosecurity measures, as well as, by vaccination (Rimler and Glisson, 1997).

Clinical signs of pasteurellosis in ratites ranged from an unapparent infection with negligible flock morbidity to episodes of per-acute mortality. Severity of pasteurellosis is influenced by climatic and nutritional factors. Clinical signs included depression and death in acute outbreaks. In chronic cases, affected birds may show arthritis, sinusitis or cellulitis. The postmortem picture was characterized by septicemia, enlargement of spleen and liver, petechial hemorrhage on the heart and generalized vascular congestion (Friend, 1987 and Tully and Shane,1996). Definitive diagnosis of pasteurellosis was made upon observing typical lesions and isolation of Pasteurella spp. from tissues .

The present study deals with a field problem showing respiratory manifestation and high mortality in ostrich farms in Egypt. Clinical, microbiological, as well as, pathological investigation were carried out in order to diagnose the causative agents responsible for this problem. Also, application of treatment trials to control the disease at ostrich farms was undertaken.

MATERIALS AND METHODS

History

Two ostrich flocks belonging to two different ostrich farms at Ismailia Governorate suffered from high morbidity and increased mortality with signs of respiratory distress, difficult breathing, anorexia, neck dropping and death, age ranged from 3-9 months. Out of 380 birds raised 109 were affected and 30 died. Erythromycin

medication had no evidence. Complete clinical signs and postmortem examination were recorded.

Sampling

At autopsy tissue smears of lungs and liver and heart blood smears were done and stained with Gram and Giemsa stains, respectively for detection of Gram negative bipolar coccobacilli. Samples were collected aseptically from liver, lung, heart, spleen, kidneys and intestine for bacteriological and pathological examination in addition to nasal swabs from diseased birds.

Bacterial isolation and identificatrion

Samples were inoculated on nutrient broth and tryptos soya broth, incubated at 37°C for 24 hrs, subcultured on blood agar, MacConky agar, EMB and incubated at 37°C for 24-48 h. Suspected colonies were picked up for morphological and biochemical characters according to Quinn *et al.* (2002).

Suspected Pasteurella colonies were subjected to indol test, urease,VP, MR, H2S production, gelatin liquefaction, ornithin decaboxylase utilization and sugar fermentation of glucose, sucrose, lactose, mannitol and maltose.

Animal pathogenicity

According to Robers *et al.* (1980), each Pasteurella isolate was inoculated in two mice intraperitonially at a dose of 0.5 ml bacterial suspension (10 -10 CFU/ml). From dead mice, heart blood was smeared and stained with Giemsa for detection of Pasteurella bipolarity and inoculated on blood agar plates for reisolation of the micro-organism.

Antibiogram study

In vitro, antibiogram test of *Pasteurella multocida* was carried out according to Quinn *et al.* (2002).

Histopathological examination

Small specimens of trachea, lungs, liver, heart, kidneys and intestine were collected from freshly dead birds and were directly fixed in 10 % neutral buffered formalin. After proper fixation, five-micron thick paraffin sections were prepared and stained with H&E (Drury and Wallington, 1980).

Field treatment

Based on antibiogram result different treatment regimens were applied. Diseased birds were grouped into 3 groups each of 20 birds and received the following antimicrobial agents in drinking water for 5 successive days.

1-Enrofloxacin (10 mg/kg b.w).

2-Amoxycillin (15mg/kg b.w) .

3-Trimethoprim/sulphamethoxazol 15 mg/kg b.w with concentration of (8mg/ml and 40 mg/ml, respectively) in drinking water, for 5 successive days. The clinical signs and mortality percentage were recoded in different groups.

RESULTS

Clinical and postmortem finding

The clinical signs of the affected ostrich birds appeared as difficult breathing, purulent nasal discharge, cough, anorexia and neck dropping. These signs continued for 2-3 days followed by complete anorexia, wing dropping and recumbency with respiratory distress and ended by death (Fig. 1). Other birds showed arthritis, lameness and diarrhea.

Postmortem examination revealed pharyngeal edema, hemorrhagic tracheitis and the trachea was occluded with bloody exudates. Pulmonary congestion with brown patches on the lungs with severe congestion and hemorrhage in the spleen and the liver were seen. Some birds showed picture of septicemia (Fig. 2) and yellowish caseous materials in the hock joint.

Bacterial isolation and identification

Initial examination of heart blood smear and liver imprint revealed presence of many Gram negative, bipolar staining short bacilli or coccobacilli micro-organisms .The result of bacteriological isolation in Tables 1&2 revealed Pasteurella spp. was recovered by (90%) 36/40 from total examined birds. Other bacteria isolated were *E.coli* 5/40 (12.5%), *Klebsiella pneumoniae* 3/40 (7.5%) and *Edwardsilla* spp. 2/40 (5%). For pathogenicity test the inoculated mice died within 24 hours with septicemia. Reisolation of the inoculated organism was done from heart blood.

The results of morphological characteristic, biochemical reactions and pathogenicity test are showen in Table 3. Isolates of Pasteurella were distinguished into *P.multocida* 30/36 (83.3 %) and *P.hemolytica*. *P.multocida* was characterized by fine translucent, non-hemolytic colonies on blood agar, failed to grow on MacConkey's agar positive for indol, glucose, sucrose, ornithin decarboxylase utilization and negative for lactose and maltose. *P.hemolytica* is a small red pin colonies on MacConkey's agar surrounded by zone of hemolysis on sheep blood agar, positive for lactose and maltose while, negative for indol, mannitol and ornithin decarboxylase utilization.

Antibiogram test

The result of in vitro antibiogram test was summarized in Table 4.

Histopathological examination

The trachea showed epithelial desquamation and sloughing with destruction of the mucous membrane (Fig.3). The submucosa was congested and infiltrated with mononuclear cells.

The lungs revealed congestion of the pulmonary blood vessels with perivascular edema, hemorrhage and mononuclear cell infiltrations. The blood vessels showed proliferation of their endothelial cells with thrombus formation. The interalveolar septae were thickened, edematus and infiltrated with leukocytes particulary neutrophils and mononuclear cells (Fig. 4).The bronchi showed mucinous degeneration and hyperplasia of the lining epithelium with hemorrhage in lamina propria and congestion of the peribronchial blood vessels (Fig. 5).The alveoli showed

presence of serofibrinous exudate with lymphocytes, macrophages and giant cells. The adjacent pulmonary parenchyma showed emphysema and collapse (Figs.6 & 7).

The liver showed vacuolar degeneration (Fig. 8) and necrosis of the hepatic cells. There were congestion of hepatic blood vessels, focal areas of hemorrhages and perivascular edema. In some cases there was also chronic cholangitis with fibrosis.

The heart revealed congestion of the myocardial blood vessels and hyaline degeneration of the cardiac muscle fibers. Edema and mononuclear cells infiltrations among degenerated cardiac muscle with focal hemorrhages areas were detected (Fig. 9).

The kidneys were congested, hemorrhagic and showed degenerative changes and necrosis in the renal tubules with mononuclear cells infiltration. Some cases showed interstitial nephritis with fibrosis in addition to cystic dilation or atrophy of renal tubules (Fig. 10).

The intestine revealed destruction of the intestinal villi with desquamation and sloughing of the epithelial lining, leukocytic infiltration in the lamina propria and submucosa with congestion of the blood vessels. The intestinal glands showed mucinous degeneration and necrosis (Fig. 11).

The results of treatment regimen (Table 5) indicated that the diseased birds which received TMP/SMX mixture at a concentration of 8 mg/ml and 40 mg/ml in drinking water developed progressive improvement in clinical signs at the end of treatment, and no more mortalities were recorded other than the two treated groups with Enrofloxacin and Amoxycillin.

DISCUSSION

Species belonging to Pasteurella demonstrate a considerable ecological diversity of the mucosal membrane of domestic and wild animals. They may be commensals or opportunistic secondary invaders of the oral cavity and upper respiratory tract (Bisgraad,1993) with the exception of *Pasteurella multocida* which is regarded as opportunistic pathogen (Muhairwa,2000).

The clinical signs observed on the affected birds were in the form of difficult breathing, nasal discharge, cough, anorexia and neck dropping. These signs lasted for 2-3 days followed by complete anorexia, recumbency, respiratory failure and ended by death. These results agreed with finding reported by El-Faki *et al.*(2002), while, Okoh (1980) reported that the death occurred suddenly without preceding clinical symptoms. Some birds developed arthritis, lameness and diarrhea. Similar results were recorded by Tully and Shane (1996). The postmortem examination revealed pharyngeal edema, hemorrhagic tracheitis and occlusion of the trachea with bloody exudates, pulmonary congestion with brown patch on the lung and general signs of septicemia (hemorrhage on the spleen, liver and pancreas). These results could be attributed to toxins originating from the causative microorganism. Similar results were reported by Okoh (1980), while El-Faki *et al.*(2002) mentioned that the lungs of most autopsied ostrich were normal and no typical lesion of pneumonia were detected except for minor congestion.

Pasteurella multocida usually enters the tissue of the birds through mucous membrane of the pharynx or upper air passages, but, it may enter through the conjunctiva or cutaneous wound (Rimler and Glisson, 1997) .
Matsumoto *et al.* (1991) showed that, following colonization in the upper respiratory tract, subsequent spread to the lungs, invasion of blood stream and septicemia usually developed. The cause of death was assumed to be endotoxin shock (Christenen and Bisgaard,1997).In contrast to septicemic nature of the acute disease , chronic form was usually characterized by localized infection and may involve hock joint (Rimler and Glisson,1997).

In the present study, it was evident that *Pasteurella multocida* was responsible for the severe pathological changes developed in lungs, which were characterized by bronchopneumonia and serofibrinous pneumonia. Similar results were described by Muhairwa (2000). Several pathological lesions were seen in liver, kidneys and heart. The occurrence of some degenerative changes in hepatic cells, shrunking glomerular

tufts, as well as, degenerative changes in the epithelium of renal tubules were attributed to infection with *P.multocida* and their circulating toxins. These results coincided with finding of Okah (1980), Dunbar *et al.*(2000)and Muhairwa (2000).

The virtual unlimited host range of *Pasteurella multocida* is indicated by reports of isolation from many wild birds (Okah, 1980). Hirsh *et al.* (1990) indicted that *P.multocida* has been predominately isolated from 100 species of wild birds.
Bacteriological isolation revealed *Pasteurella* spp 936/40), *E.coli* (5/40), *Klebsiella pneumoniae* (3/40) and *Edwardsiella* spp (2/40). According to the results of growth charctersitcs, mice pathogenicity and biochemical reactions, isolates of *Pasteurella* spp. were distinguished into *P.mulocida* and P. *hemolytica.* Similar differences between *P.mulocida* and *P. hemolytica* in growth characterisitics and biochemical reactions were reported by Backall *et al.* (1995),Rimler and Glisson(1997) and Quinen *et al.(*2002).
Also, Rimeler and Glisson (1997) stated that the microbial isolation is the gold standard for the identification of an isolate, but morphological feature of *P.mulocida* is not characteristic enough to differentiate from other Gram negative Coccobacilli ; therefore some key biochemical features include positive indol reaction and ornithine decarboxylase activity in addition to mice inoculation are used in identification and differentiation of Pasteurella strains.

The isolated *Pateurella multocida* organism from dead and diseased birds was highly pathogenic for mice (killed mice within 18-24 hours after injection).In this context, Woolcock and Collins (1975), reported that small doses of *P.multocida* were sufficient to kill a mice.

Okah (1980) and El-Faki *et al.*(2002) reported the isolation of *Pateurella multocida* from septicemic cases in ostrich .The results indicated the isolation of *P.hemolytica* in combination with *P.mulocida.* This is because *Pasteurella hemolytica* is mostly secondary pathogen (Hocking and Pettit,1974) and closely related to isolates of *P.multocida* recoverd from diseased poultry (Heddleston,1975).Rabab (2003) isolated P.hemolytica from ostrich farms in Ismailia governorate, while, Amal and Mohamed

(2000) isolated *E.coli, Klebesiella* spp and *Edwardsiella* spp. from cases of ostrich which agreed with the present study.

Antibiogram test for isolated *P.multocida* revealed their susceptibility to trimethoprim/ sulphamethoxazol, ceftiofur and amoxicillin, less sensitive to enrofloxacin and Tetracycline and resistant to lincospectin and danofloxacin.This result agreed with Raemdenk *et al.* (1992) and El-Faki *et al.* (2002). Lin *et al.* (2001) found that *P.mulocida* isolated from chicken was highly sensitive to Neomycin and chloramphenicol.Also, Jonas *et al.*(2001) stated that all examined *P.mulocida* strains were susceptible to trimethoprim and ampicillin and less sensitive to enrofloxacin.

Application of treatment regimens revealed that a mixture of trimethoprime /sulphamethoxazol at a dose rate of (8 mg/ml, 40 mg/ml) in drinking water for 5 successive days was effective for controlling mortality rate and induced progressive improvement in clinical signs than application of enrofloxacin and amoxycillin.Such results completely agreed with those of El-Faki *et al.*(2002) who found that application of trimethoprim/sulphamethoxazol controlled the outbreaks of pasteurellosis in ostrich and no further losses were reported at the end of treatment .

Rimler and Glisson (1997) reported that sulphonamides have been employed in treatment of pasteurellosis in both experimental and naturally occurring outbreaks. The main advantage of sulphonamides is their bacteriostatic action to control localized infection and toxic effect on birds.

It is clear from the present findings that *Pasteurella mulocida* seemed to be the main cause of losses. This conclusion is reached from isolation of *P.multocida* in a significant percentage from diseased and freshly dead birds. History of failure to control losses after erythromycin medication was driven from improper diagnosis, so, caution is drawn to the use of medication without definitive diagnosis by microbial isolation followed by antibiotic sensitivity test.

Table 1. Incidence of bacterial isolation from total examind ostriches.

Bacterial pathogens	Incidence of isolation n=40	
	No.	%
Pasteurella spp.	36	90
Escherichia coli	5	12.5
Klebsiella pneumoniae	3	7.5
Edwardsiella spp.	2	5

Table 2. Incidence of bacterial isolation from freshly dead and diseased ostriches

Bacterial isolates	Freshly died n = 30		Diseased birds n = 10	
	No.	%	No.	%
Pateurella mulocida	27	90	3	30
Pasteurella hemolytica	3	10	3	30
E.coli	5	16.6	-	-
Klebsiella pneumoniae	3	10	-	-
Edwardsiella spp.	2	6.66	-	-

Table3. Results of mice pathogencity, hemolytic activity and biochemical reactions of isolated Pasteurellae.

Farm	*Age*	*No. of examined*	*No.of isolates*	*Growth on mac agar*	*Pathogenicity to mice*	*Hemolysis On Blood agar*	*Indol*	*urease*	*Ornithin decrboxlas*	*Sugar fermentation*				
										Glucose	*Sucrose*	*Maltose*	*manniol*	*Lactose*
1	3-5 months	18	16	4	12	4	12	-	12	16	16	4	15	4
2	4-9 months	22	20	2	18	2	18	-	18	20	20	2	19	2
Total		40	36	6	30	6	30	-	30	36	36	6	34	6

Table 4. Antibiogram study of isolated *Pasteurella multocida*.

Antimicrobial	*Disc concenteration* µg	*Degree of sensitivity n = 30*			
		S		*R*	
		No.	*%*	*No.*	*%*
Amoxycillin	10 µg	30	100	-	-
Ampicillin	10 µg	28	93.33	2	6.67
Ceftiofur	10 µg	30	100	-	-
Danofloxacin	30 µg	-	-	30	100
Chloramephenicol	10 µg	9	30	21	70
Erythromycin	15 µg	21	70	9	30
Enrofloxacin	10 µg	24	80	6	20
Gentamycin	10 µg	27	90	3	10
Lincospectin	150 µg	-	-	30	100
Penicillin	10 µg	26	66.66	4	13.3
Trimethoprim/sulphamethoxazol	25 µg	30	100	-	-
Tetracycline	30 µg	24	80	6	20

Table 5. Treatment regimen for affected ostrich by different antimicrobials.

Treatments	No.of birds	Respiratory signs		Mortality	
		No.	%	No.	%
Enrofloxacin	20	4	25	2	10
Amoxycillin	20	2	10	1	5
TMP/SMX	20	1	5	-	-

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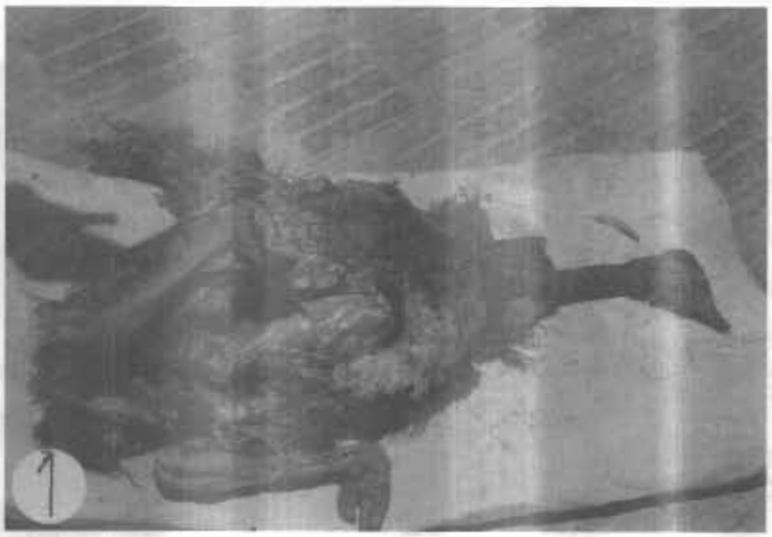
List of figures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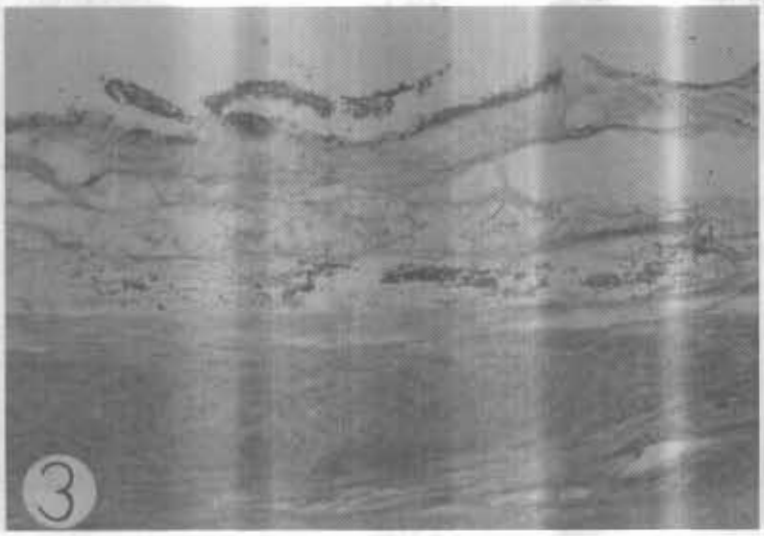
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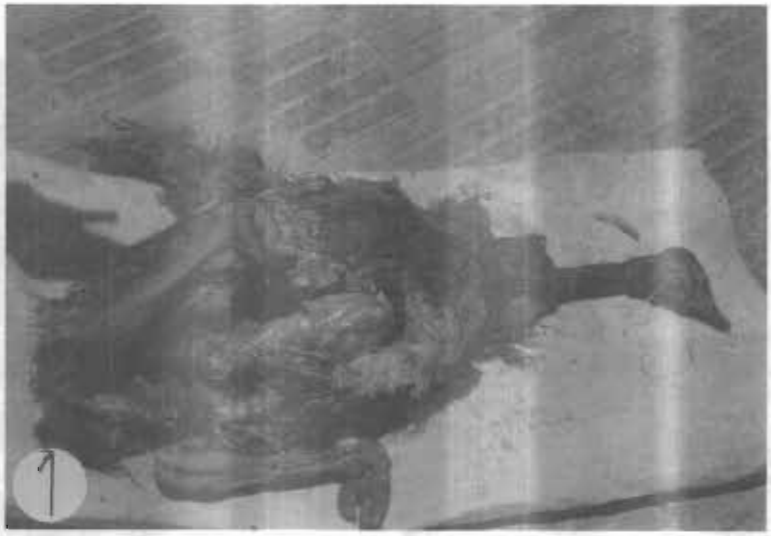
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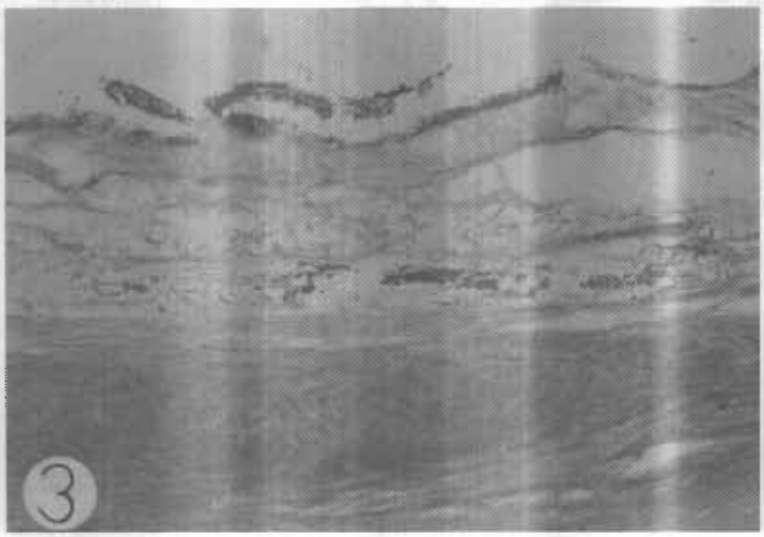
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الباستيرلا في النعام

سهير يوسف محمد ، محمد كمال مرسي دسوقي

معهد بحوث صحة الحيوان – مركز البحوث الزراعية – وزارة الزراعة الدقى – جيزة – مصر .

أجريت هذه الدراسة على مزرعتين من مزارع النعام التى تعاني من نسبة إصابة عالية و زيادة في معدل النفوق مع ظهور أعراض تنفسية مختلفة مثل صعوبة التنفس مع افرازات أنفية و كحة بالاضافة الى حالات نفوق. وقد تم أجراء الصفة التشريحية و الفحص البكتيريولوجي مع أجراء اختبار الحساسية بالمضادات الحيوية للمعزولات من ميكروب الباستيرلا ملتوسيدا للوقوف على العلاج المناسب. . وقد تم عزل ميكروب الباستيرلا ملتوسيدا بمعدل ٩٠ % من إجمالي الحالات التى تم فحصها و تم تنصنيفه بيوكميائيا وباستخدام الضراوة وذلك بحقنه في فئران التجارب . كما تم عزل ميكروب الباستيرلا هيمولتيكا وميكروب الكلابسيلا والميكروب القولونى وميكروب الادوارد سيلا بنسب متفاوتة. وقد أفاد الفحص الهيستوباثولوجي وجود تغيرات باثولوجية شديدة بالرئتين مع التهاب البلورا ووجود التهاب رئوي صديدي تليفي مع ظهور تغيرات انحلالية مختلفة بالاعضاء الداخلية. وقد أشارت نتائج اختبار الحساسية لميكروب الباستيرلاملتوسيدا حساسيتها الى كل من التراي ميثوبريم / السلفاميثوكسازول و السفيتوفور و الاموكسيسيلين بنسبة (١٠٠ %) و اقل حساسية لكل من التتراسيكيلين (٩٣%) والانروفلوكساسين (٨٠%) . طبقا لنتائج اختبار الحساسية تم تطبيق نظام علاجي باستخدام خليط من تراي ميثوبريم والسلفاميثوكسازول بتركيزات ٨مجم/مل ، ٤٠مجم/مل على التوالي وبجرعة ١٥مجم/كجم من وزن الطائر وذلك عن طريق مياة الشرب لمدة خمسة أيام متتالية .وقد وجد أن هذة الطريقة فعالة في تحسين الأعراض الظاهرية على الطائر و تقليل نسبة النفوق.