Dept. of Pathology,
Fac. of Vet. Med., Moshtohour, Zagazig University.
Head of Dept. Dr. A.A. Nagi.

CLINICO-PATHOLOGICAL AND BACTERIOLOGICAL STUDIES ON AVIAN PASTEURELLOSIS

(With 6 Figures)

By

A.A. NAGI, M.S. YOUSSEF*, S. MOUSA and A.H. BAYOUMI***

(Received at 10/10/1989)

دراسات إكلينيكية ، باثولوجيـة وبكتريولوجية عن كوليرا الطيـــور

عبد الرحيم ناجي ، محمد صلاح يوسف ، صلاح موسى ، عبداللطيف بيومي

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

*: Dept. of Pathology, Fac. of Vet. Medicine, Assiut University.
**: Dept. of Poultry Diseases, Fac. of Vet. Medicine, Assiut University.

and to a lesser extent in the ovaries and joints (OLSON and McCUNE, 1968 and MOUSA et al., 1988).

In the last two years, several outbreaks with high morbidity and mortality rates were observed in breeder poultry farms. The aim of the present work was to describe the clinico-pathological alterations of the disease, and to isolate and serologically identify the causative organism.

MATERIALS and METHODS

In this investigation, 320 freshly dead and moribund cases of adult chickens (6-10 months) were subjected to post-mortem, bacteriological and histopathological examinations.

Careful dissection was carried out and fresh tissue specimens were taken from the skin, liver, heart, trachea, lungs and intestines. All materials were fixed in 10% neutral buffered formalin and processed by conventional techniques for histopathlogical examination. Sections were cut at 5 micron thick and stained with haematoxylin and eosin (H & E).

For isolation and identification of the causative organism, blood films as well as smears from the liver and wattle-exudate were obtained, stained with Giemsa, and examined. Cultures from heart blood, liver and wattle-exudate were made on tryptose broth and blood agar. Final identification was carried out according to the methods described by NAMIOKA and MURATA (1961) and HEDDLESTON (1975).

RESULTS

Clinical signs :

The affected flocks in all outbreaks showed high mortality rate ranged from 20-25%. In many cases, death was the first indication of the disease. Otherwise, clinical signs including diarrhea, increased respiratory rate, ruffled feathers and cyanosis of comb and wattles were noticed. In the chronic persisting infections, nasal discharge, respiratory distress and conjunctivitis were the most prominent clinical signs. Lamness was not infrequently observed.

Gross findings :

On necropsy, the gross lesions of the all affected birds were nearly constant and more or less similar in type and severity. In the acute cases, circulatory disturbances were pronounced and exhibited general vascular congestion, peticheal and ecchymotic haemorrhages on the pectoral muscles; coronary fat; serosal and mucosal membranes. The liver was enlarged, congested and infrequently revealed multiple greyish-white foci. The long standing cases showed severe oedema of the head, comb and wattles (Fig. 1). Congestion of the ovules, lungs and intestines, and swelling of the hock joints could be also observed. Dirty yellowish, thick mucoid exudate was oftently present in the upper respiratory passages.

Histopathological findings :

In the acute cases, the microscopical alterations were only restricted to the liver. Congestion of the sinusoids as well as heterophlic cellular infiltrations were observed (Fig. 2). while in chronic cases, degeneration of the hepatocytes was also noticed. Moreover, few minute foci of coagulative necrosis were demonstrated (Fig.3).

In the trachea, a pseudodiphtheritic-like membrane of mucoid exudate was irregularly extended on the epithelial surface. The lungs of some cases revealed few pneumonic foci. The heart showed severe congestion of the coronary vascular ramifications, and perivascular and interstitial oedema (Fig. 4).

The small intestine exhibited necrobiotic changes of the villus epithelium together with prominent heterophilic cellular infiltrations in the mucosa and submucosa (Fig. 5).

In the chronic persisting cases, the subcutis of the neck and wattles was hyperaemic and oedematous (Fig. 6 a,b).

Bacteriological findings :

Blood and tissue smears showed the presence of a bipolar coccobacilli inbetween the cellular elements. Bacteriological examinations revealed the isoaltion of twenty-five isoaltes of Pasteurella multocida which were non-haemolytic on blood agar, and lethal to mice and rabbits. The isolates were serologically identified as 5 : A (11 isolates) and 8 : A (14 isolates).

DISCUSSION

In the present work, the clinical signs and lesions observed in the affected flocks pointed to a typical course of infection with an acute sudden onset fated with a chronic localized form of fowl cholera. similar signs and lesions were also described by RHODES and RIMLER (1984).

Microscopically, the acute cases were characterized by pronounced circulatory disturbances and heterophilic infiltrations. Degeneration and necrosis seen in the liver of chronic cases could be attributed to the direct and indirect actions of the causative organism. These findings are quite similar to those described by COHRS (1966). Swelling of the head, wattles and joints were the prominent features observed in the long standing cases. SIRBU et al. (1971) reported that such lesions are common sequelae of acute infection specially after chemotherapy with bacteriostatic drugs. However, localization of Pasteurella multocida organisms in the subcutaneous tissue and joints could be responsible for such chronic lesions in these organs.

As shown in the present investigation, our result revealed that all isolates possessed the specific biochemical and cultural characteristics of Pasteurella multocida described by HEDDLESTON (1975). The isolates were typed as 5 : A and, 8 : A serotypes.

Similar results were obtained by NAMIOKA (1970) who recorded that Pasteurella multocida 5 : A, 8 : A and 9 : A were the only serotypes capable of producing fowl cholera. In addition, CARTER and BAIN (1960), EL-MONGY (1977) and MOUSA et al. (1988) concluded that type : A was highly pathogenic and the most prevalent type affecting avian species.

REFERENCES

Carter, J.R. and Bain, R.V.S. (1960): Pasteurellosis (Pasteurella multocida): A review stressing recent developments. Vet. Rev. and Annot. J.: 105-128.

Cohrs, P. (1966): Text book of special pathological anatomy of domestic animals. English ed., Pergamon press, Oxfored.

El-Mongy, F.A.A. (1977): Studies on duck pasteurellosis. Thesis presented in the Fac. of Vet. Med., Cairo University.

Heddleston, K.L. (1962): Studies on pasteurellosis: V- Two immunogenic types of P. multocida associated with fowl cholera. Avian Dis., 6: 315-321.

Heddleston, K.L. (1975): Pasteurellosis. In Isolation and Identification of Avian Pathogens. Am. Assoc. of Avian Pathologists. Texas A. and M. University College Station.

Mousa, S.; Soliman, A.; Ibrahim, A.; Nahed Gad and Shehata, M. (1988): Some studies on fowl cholera in Upper Egypt. Third Sci. Cong., Fac. of Vet. Med., Assiut University, 20-22 November 1988: 343.

Namioka, S. (1970): Antigenic analysis of Pasteurella multocida. Nat. Inst. Anim. Health. Quart, Vol.: 10, Sluppl. 97-108.

Namioka, S. and Murata, M. (1961): Serological studies on Pasteurella multocida: I-A simplified method for capsule typing of the organism. Cornell Vet., 51: 498.

Olson, L.D. and McCune, C. (1968): Studies on avian pasteurellosis. Am. J. Vet. Res., 29: 1665.

Rhodes, K.R. and Rimler, R.B. (1984): Avian pasteurellosis in Diseases of Poultry. 8th ed., Iowa State Univ. Press, Ames Iowa, U.S.A.

Sirbu, S.; Paunescu, G. and Gogoasa, V. (1971): Postmortem, Histological and haematological investigations of fowls from pasteurellosis outbreaks. Revista de 200 techni technie, Si., Medicina Veterinara, 21: 64-70.

DESCRIPTION OF FIGURES

Fig. 1: A hen showing severe swelling of the wattles.

Fig. 2: Liver showing congestion and heterophilic infiltrations. H & E stain, X 160.

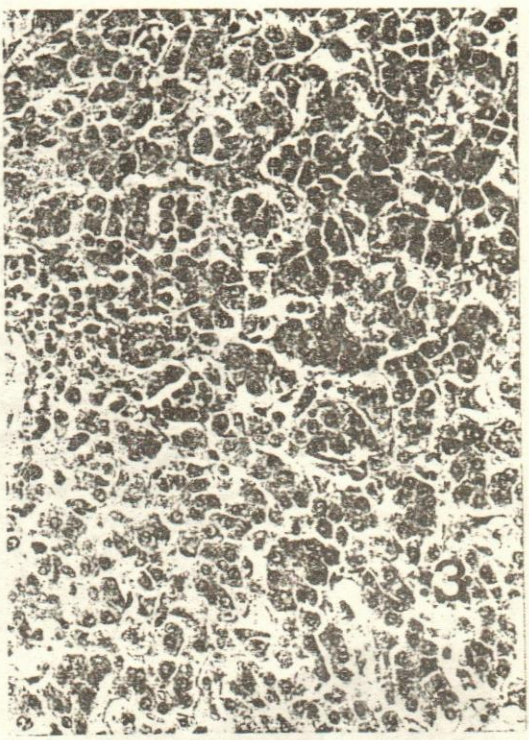
Fig. 3: Liver showing focal coagulative necrosis. H & E stain, X 250.

Fig. 4: Heart showing perivascular and interstitial oedema. H & E stain, X 250.

Fig. 5: Small intestine showing necrobiotic changes of the villus epithelium together with heterophilic infiltrations. H & E stain, X 250.

Fig. 6: a,b: Skin showing subcutaneous oedema. H & E stain, X 160, X 250.

PATHOLOGY & BACTERIOLOGY OF PASTEURELLOSIS



5

6(a)

6 (b)