Dept. of Pathology,
Faculty of Vet. Med., Assiut University,
Head of Dept. Prof. Dr. H.A. Salem.

EFFECT OF ESCHERICHIA COLI ON PASTEURELLA MULTOCIDA INFECTION IN CHICKENS

(With One Table & 5 Figs.)

By
A.Z. MAHMOUD and M.K. IBRAHIM

(Received at 8/5/1988)

تأثير ميكروب الاشرشياكولاى على الاصابة بميكروب البستريلا في الفراخ

محمود عبدالظاهر، محمد خيرى

استخدمت ٦٠ من البدارى لهذا البحث. وقد قسمت الى ثلاثة مجموعات حقنت المجموعة الأولى بميكروب البستريلا والثالثة بميكروب الاشرشيا كولاى أما المجموعة الثانية فحقنت بالاثنين معا. نفقت بدارى المجموعة الأولى نتيجة للاصابة الحادة فتسمم الدم ونفق فقط ٣٠٪ من المجموعة الثانية أما المجموعة الثالثة فكانت الاصابة مزمنة ولم ينفق شيء منها. وقد استخلص من هذه النتائج أن الحقن بميكروب الاشرشيا كولاى منع ٧٠٪ من النفوق في البدارى التي حقنت بميكروب البستريلا.

SUMMARY

60 broiler checkens were used for this study. They were divided equally into three groups. They were experimentally infected with pasteurella multocida, E.Coli and Pasteurella multocida and E.Coli respectively. All chickens in the first group infected with P. multocida died 24-48 hours post infection. Pathological studies in this group revealed acute septicemic lesions. In the second group infected with E.Coli and P.multocida, the lesions tend to be subacute or chronic. Only 30% of chickens in this group died from acute pasteurellosis. In all chickens of the third group (infected with E.Coli only) chronic proliferative inflammation was observed in the lung and serous membranes. In conclusion, E.Coli infection protected 70% of chickens from acute septicemic pasteurellosis and prevent their sudden death.

INTRODUCTION

A complicated infection with pasteurella multocida and escherichia coli were frequently observed in broiler chickens presented to the department of poultry science, Auburn University Auburn-Alabama U.S.A. (MAHMOUD and MORA, 1986). Pathological studies of this chicken revealed chronic lesions characteristic for Pasteurella multocida infection. Beside these lesions a granulomatus proliferative inflammation characteristic for E.Coli infection was observed in the serous membranes and internal organs. Apparently pasteurella multocida infection occurred First, while E.Coli infection was secondary. This study was performed to throw some light on the reverse effect i.e the effect of E.Coli on pasteurella multocida infection in chickens and to describe the pathological picture induced by this mixed infections.

MATERIAL and METHODS

This study was performed on 60 broiler chickens 6 weeks of age. The chickens were divided into three equall groups each contained 20 birds. The first group was injected with pasteurella multocida. The second group was infected with E.Coli and pasteurella multocida. The third group was infected with E.Coli only (Table 1). In addition 10 chickens were used as a control.

The culture of E.Coli (strain R. 12) had been isolated from an outbreak of colibacillosis in chickens. Pasteurella multocida was isolated from an outbreak of pasteurullosis of chickens in the department of poultry science Auburn University, Auburn Alabama USA. All infected chickens were put under observation and rationed libtum. All chickens in the first group died 24-48 hours post infection. Six chicken from the second group died on the second, third and fourth days post infection (two birds every day). No mortality was observed among chickens of the third group. All the remaining chickens were killed after 15 days post infections. Samples from different organs inculding, liver, intestine peritoneium, spleen, lung and kidney were fixed in Bouin's fluid then processed. Paraffin sections were made at a thickness of 4-6 micron, stained by H & E and examined.

RESULTS

Gross studies:

Examination of dead chickens from the first group (died 24-48 hours post infection) revealed signs of septicemia. A large amount of strow coloured fluid were found in the peritoneal cavity. The blood vesseles of the peritoneum and serosal surfaces of the intestine were prominently congested. Liver, Spleen, Kidneys and Lungs were swollen oedematus and dark red in colour. The heart was flabby and filled with clotted blood. Petecheal hemorrhages were always seen on the serosal surfaces of the intestine and heart.

Gross examination of organs from chickens died with mixed infection revealed slight enlargment and congestion. The liver contained whitish spots. The spleen was also enlarged and congested. The lung contained dark red hepatized areas.

Chickens injected with E.Coli in the third group. Showed thickening of serous membranes inculding those of the peritoneum, pericardium and pleura. The liver was enlarged and congested, contained whitish spots. The spleen was also enlarged and the lung showed focal areas of red hepatization. The kidneys were also enlarged.

Micromorphological studies:

A. First group:

The liver showed prominent congestion and thrombosis of the portal blood vesseles and central veins.

Lymphoid cell infiltrations were seen in the portal tract and around the central veins. Focal areas of hydropic degeneration of the liver cells were not infrequently observed. Micromorphological changes in the lung consisted of congestion, oedema and hemorrhages in some areas. The spleen showed congestion of the red pulp. The intestinal mucosa showed capillary congestion and desqumation of its epithelium.

B. Second group:

Examination of organs from chickens died with mixed infection revealed that the liver contained multiple small focal areas of coagulative necrosis, which sometimes involve only few liver cells. Focal areas of hydropic degeneration were seen. Large areas of cellular reaction were observed in the portal tract and around the central vein (Fig. 1). The reacting cells were macrophages, lymphoid and esinophil cells. The liver from birds which were killed 15 days post infection showed small focal areas of subacute or chronic hepatitis, the reacting cells were macrophages, lymphoid and esinophil cells.

The intestine from chickens died with mixed infection revealed extensive necrosis and desqumation of the enterocytes with cellular reaction. These changes were spread to involve large segments of both small and large intestine. The intestine from killed chickens showed focal areas of macrophages and lymphoid cells infiltrations of the mucosa. Hyperplasia of the enterocytes was sometimes seen in some areas. The spleen from dead chickens showed lymphoid hyperplasia and moderate histocytic prolfferation, while the spleen from killed chickens showed only prominent histocytic proliferation (Fig. 2). The lung from dead chickens showed focal areas of pneumonia. The reacting cells were of heterophil, macrophages and lymphocytic type. Oedema of the lung was frequently observed.

The lung from killed chickens showed minute focal areas of subacute or chronic pneumonia. The reacting cells were lymphoid cells and macrophages. In the kidneys of dead chickens the tubular epithelium showed focal areas of hydropic degeneration and the interstitium showed macrophage cell infiltrations. While in killed chickens the latter was only observed.

C. Third group:

Examination of the organs from this group showed that the lesions were of chronic type.

The liver showed large focal areas of cellular reaction which were usually seen in the portal tract and around the central veins. The reacting cells were macrophages, lymphoid cells and abundant population of esinophil cells. The hepatic cells in the area of cellular reaction were completely destroyed. Focal area of hepatic cell degeneration were commonly observed. Necrotic process which involve several hepatic cells were frequently seen. The intestinal mucosa showed severe hyperplastic changes (Fig. 3). The mucosa was drown into folds and consists of several rows of epithelial cells. The hyperplastic epithelium sometimes showed necrotic changes of frank coagulative type. The connective tissue core of the villi showed light lymphoid and heterophil cells infiltration. The peritoneum, pleura and pericardium were thickend by fibroblastic proliferation. They were also infiltrated with lymphoid and epithelioid cells. Giant cells were also observed in the intestinal peritoneum (Fig. 4). The lung showed focal areas of granulomatus pneumonia, along with oedema and congestion. In the pneumonic areas extensive proliferation of epithelioid, lymphoid and giant cells were observed (Fig. 5). Necrotic changes may involve the giant cells whose nucleus fused to form continuous line. The spleen showed lymphoid cell hyperplasia, the germinal centre of the white pulp consists of blast cells. The kidneys showed focal areas of lymphoid and macrophages cells reaction in the interstitial connective tissues. Light mesangial cells proliferation and evidence of vaccuolar degeneration of the renal tubular epithelium were also observed.

DISCUSSION

In this study all chickens infected with pasteurella multocida via the nasal cleft died with a symptoms of septicemia (RUNNELL, et al. 1965). Only 30% of chickens in the second group died on the second, third and fourth day post infection. In these chickens, E.Coli infection somewhat delayed the death of this chickens, and decrease the severity of the acute disease. Lesions in these chickens were of three types. The first type represented by alterative changes observed in the liver, intestine and kidneys. The second type of lesions represented by inflammatory changes e.g. Local pneumonia, pericarditis pleuritis and peritonitis. These two type of lesions were due to the direct effect of both pasteurella multocida and E.Coli (RUNNELLES, et al. 1965; GROSS, 1961; GROSS, 1966). The third type of lesions represented by focal areas of lymphoid and macrophages cells infiltratin of the liver along with lymphoid and histocytic cells proliferation in the spleen. These changes in our opinion represented an immune defence reaction of the body against this mixed bacterial infection (ALI, 1985 and TAYLOR and BURROUNHS, 1973). In chickens infected with both organisms and did not die but killed 15 days post infection (70%), adistinct immune response was observed in the spleen (prominent lymphoid and histocytic cells proliferation). This immune response in our opinion may be responsible for the protection of these chickens from acute illness and prevented their sudden death.

Apparently this immune response developed as a result of multiple injection by small doses of E.Coli before administration of pasteurella multocida. Lesions in the third group consists of proliferative and granulomatus inflammatory changes in the lung and serous membranes. Multiple coli granulomas rich in esinophil cells were also observed in the liver (HAMILTON and CONARD, 1958) and (HJARRE and WRAMBY, 1945). Enteritis caused by E.Coli is important in humans, calves ans swine (GROSS, 1983). While there are few reports of enteritis in poultry in which E.Coli has been suggested as playing a part. Enough conclusive research to indicate that E.Coli an etiological agent for enteritis in chickens, (NAGI and RAGGI, 1972). Histopathological picture of the intestine from chickens of the third group proved that E.Coli infection could induce proliferative type of enteritis.

From this study we can concluded that E.Coli infection could protect 70% chickens from acute septicemic pasterurellosis and prevent their sudden death. We can also concluded that E.Coli infection could induce proliferative enteritis in experimentally infected chickens.

REFERENCES

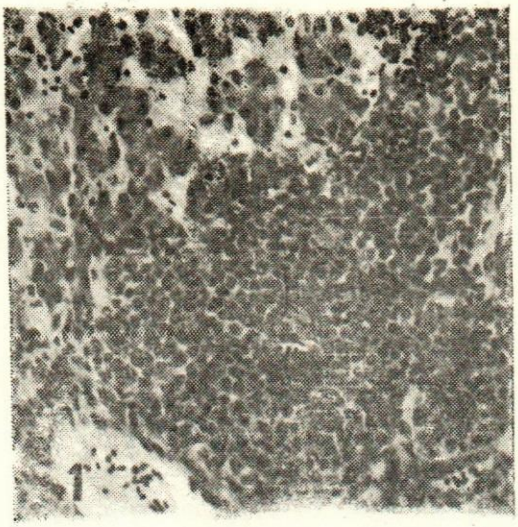
Ali, S.R. (1985): Histopathological and serum electrophoretic studies on experimental Aspergillosis in pigeons. Thesis, M.V.Sc. Faculty of Vet. Med., Assiut University.

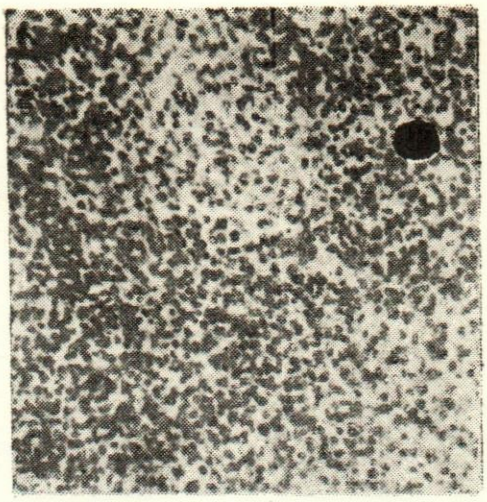
Gross, W.B. (1961): The development of air sac disease. Avian Dis., 5. 431-439.

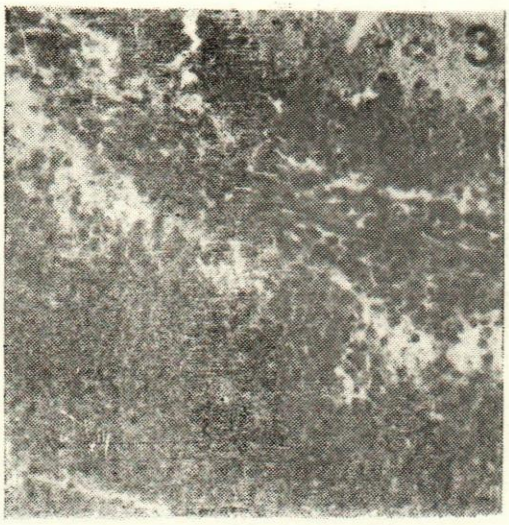
Gross, W.B. (1966): Cited after Hofstad. M.S. (1984): Diseases of poultry. 8th Ed. Iowa state University Press, Ames, Iowa, USA.

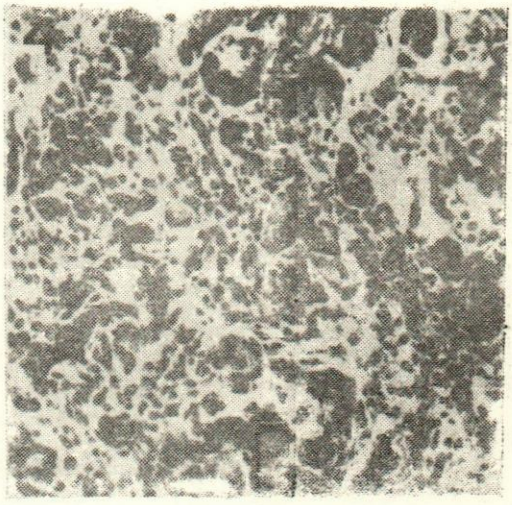
Gross, W.B. (1983): Cited after Hofstad M.S. (1984): Diseases of poultry 8th Ed. Iowa state Univ. Press, Ames, Iowa, USA.

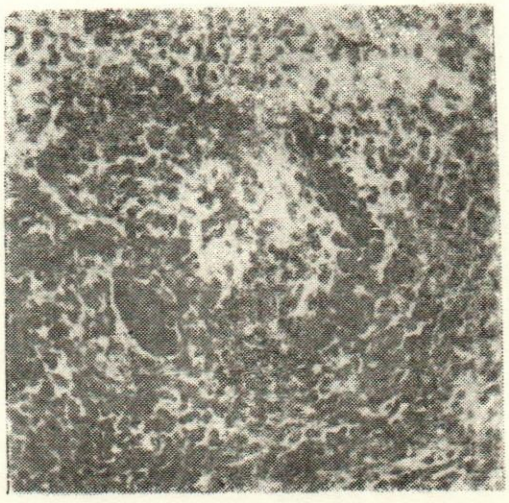
Hamilton, C.M. and R.D. Conard (1958): Extreme mortality in Hjarre's disease (coligranuloma) in chickens. J. Am. Vet. Med. Assoc. 132, 84-85.

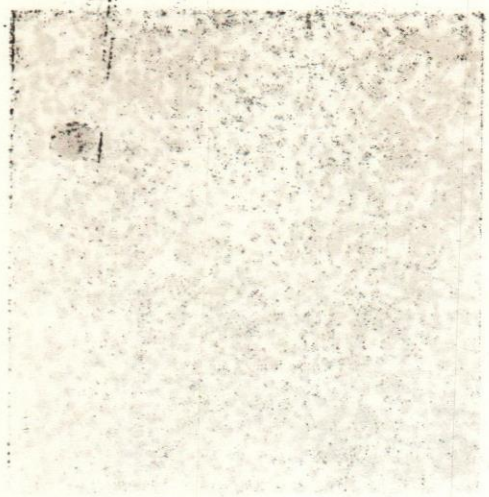

1

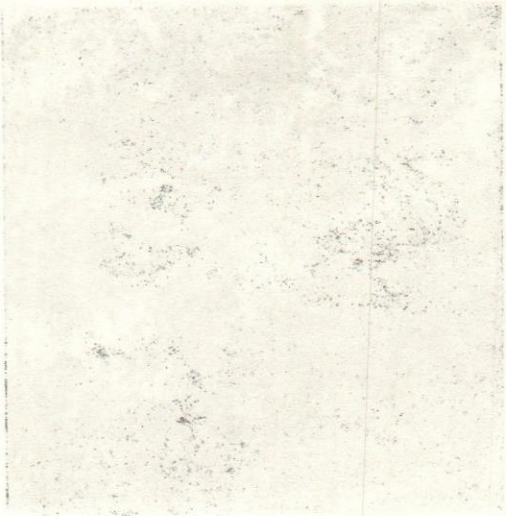


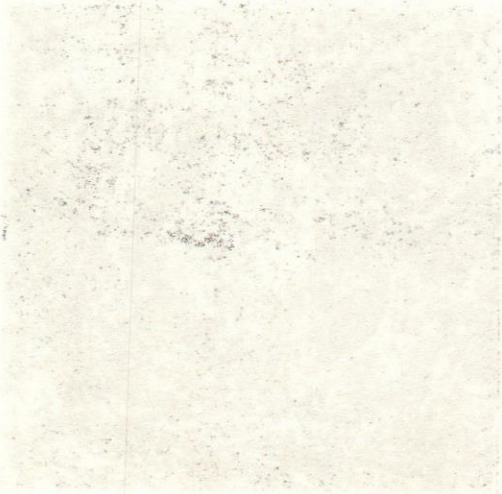

3











Mahmoud, A.Z. and F.C. Mora (1986): Analysis of lesions induced by Escherichia Coli secondary infection in chickens previously vaccinanted with live attenuated strain of pasteurella multocide. Under Press.

Hjarre, A. and G. Wramby (1945): Cited after Hofstad M.S. (1984): Diseases of poultry 8th Ed. Iowa state University Press, Ames. Iowa, USA.

Nagi, M.S. and I.G. Raggi (1972): Importance to "Air Sac" disease of water supplies contaminated with pathogenic Escherichia Coli. Avian Dis. 15: 718-723.

Runnells, R.A.; Monlux, W.S. and Monlux, A.W. (1965): Principles of Vet. Pathology seventh Ed. Ames. Iowa, USA.

Taylor, J.J. and Burrounhs, E.J. (1973): Experimental avian Aspergillosis. Myco pathologia and Mycologic Applicate, 51, 131-141.

LEGENDS OF FIGURES

Fig. (1): Cellular reaction in the liver (H & E, 25 X 12.5).
Fig. (2): Histocytic proliferation in the spleen (H & E, 25 X 12.5).
Fig. (3): Hyperplasia of enterocytes (H & E, 25 X 12.5).
Fig. (4): Granulomatous peritonitis (H & E, 25 X 12.5).
Fig. (5): Granulomatous pneumonia (H & E, 25 X 12.5).

Table (1)
Showing time and dose of infection in the different groups

Time of infection	First group	Second group	Third group
First day	-	0.1 ml of E.Coli culture suspension in the wing. vein.	0.1 ml of E.Coli culture suspension in the wing vein.
Second day	-	0.1 ml of E.Coli culture suspension in the wing vein	0.1 ml of E.Coli culture suspension in the wing vein.
Third day	0.5 ml of P.multocida culture suspension in the palatin cleft.	0.5 ml of E.Coli culture suspension in the wing vein.	0.5 ml of E.Coli culture suspension in the wing vein.
Fourth day		0.5 ml of P.multocida culture suspension in the palatin cleft.	