قسم : الباثولوجيـــــــــا .
كلية : الطب البيطرى ـ جامعة أسيوط.
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# الصوره الباثولوجيه في الكتاكيت المعدية تجريبيا

## بعترة ( B 17 ) K 72 : 124 0

## من الميكروب القولونـــــي

علام نفادى ، عوض ابراهيم* ، مصطفى شحاتـــــــه*

أحدثت العدوى التجريبيه لعدد ٣٠ كتكوت بدارى عمر ٦ أسابيع عن طريق الفم استخدام عترة ( B 17 ) K 72 : 124 0 من الميكروب القولوني .

تم وضع الكتاكيت المعدية تحت الاختبار لمدة ٦ أسابيع وقد تم اجـــــراء الفحوص الاكلينيكية وتسجيل الصفات التشريحية سواء العينيه أو الميكروسكوبيـه .

الطيور التي ماتت أثناء الاصابه الحاده بالمرض أظهرت صوره التســـــمم الدموى بينما تلك التي أعدمت بعد ٦ أسابيع من العدوى أوضحت وجـــود التهابات تكاثريه في الأمعاء والكبـــــد .

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# *PATHOLOGICAL PICTURE OF E.COLI SEROGROUP 0124: k72 (B17) IN EXPERIMENTALLY INFECTED CHICKS*

*(With 10 Figs.)*

*BY*
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## *SUMMARY*

Experimental infection with E.coli, serogroup 0124:k72 (B17) was carried out on 30 six-weeks-old broilers via the oral route. The chicks were kept under observation for six weeks. Clinical observation, gross pathology and histopathological studies were conducted. Dead birds in acute stage of infection revealed features of septicaemia, while those sacrificed six weeks post-infection showed picture of productive reaction in the intestine and liver.

## *INTRODUCTION*

Coli infection of chickens is considered as one of the most serious problems affecting the poultry industry. It includes several forms of infections, such as colibacillosis which is characterized by septicaemia, pericarditis, air-sacculitis, perihepatitis and peritonitis (GROSS, 1958; HARRY, 1964 and PIERCY and WEST, 1976). While coligranuloma is a chronic disease characterized by granulomatous reaction of intestine and liver (HJARRE and WRAMBY, 1945 and HAMILTON and CONRAD, 1958).

SOJKA and CARNAGHAN (1961) and HARRY and HEMSLY (1965) stated that, field outbreaks of avian colibacillosis were associated with few E.coli serotypes. Not only the pathogenic strains of E.coli, but also the endotoxins could induce signs of the disease on affected chickens (BUTLER, CURTIS, and HARRY, 1977 and ADLER and DAMASSA, 1979).

Most of the available literatures on avian colibacillosis deals with trials for isolation of the causative organisms, gross pathology, and control programes (PIERCY and WEST, 1976). Recently the problem of E.coli infections was studied in Egypt by IBRAHIM and SHAHATA (1982), who isolated three serogroups 044:K74 (L), 0124:K72 (B17) and 0127:K70 (B15).

The present work was designed to study the pathological changes in experimentally infected broiler chickens with E.coli serogroup 0124:K72 (B17).

## *MATERIAL and METHODS*

**The organism:** E.coli serogroup 0124:K72 (B17) supplied by Ibrahim and Shahata = Poultry Deseases Dep., Fac. of Vet. Med. Assiut Uni. Egypt. = was used in the present study.

**Experimental birds:** Fourty 6 weeks broiler Dokki 4 chicks proved to be free from coli infection by trials for isolation.

**Methods:** Chicks fed by nonmedicated ration and water add libitium. Ten chicks were considered as a control group. 30 chicks were inoculated per os by 24 hour broth culture of the E.coli strain "20x10" organisms/bird. The chickens were kept under observation for 6 weeks post-infection.

Clinical observation and post mortum examination of both dead and sacrificed birds were carred out. Specimens from intestine, liver, kidney, heart, lung, spleen and brain were taken and fixed in neutral buffer formallin and embeded in paraffin wax. Sections in thickness of 4-6 U were done. Different stains such as heamatoxylin and eosin, masson trichrome and PAS reaction were done.

## *RESULTS*

**Clinical symptoms:** Loss of appetite, diarrhea, depression and congestion of the mucous membrans were observed three days postinoculation on all birds. These symptoms persisted for 4 days. Eight birds died within this period. While the symptoms in surviving birds gradually disappeared and returned to normal condition 7 days later.

**Gross pathology:** The gross pathological changes which were noticed in dead birds during the acute stage were severe congestion of the intestine, liver, lungs, kidneys, heart, spleen and brain meninges. Also a moderate serous exudate in the pericardial and air sacs could be noticed. While the sacrificed birds in chronic stage (6 weeks post-infection) showed only palness of the liver and kidneys and thickening of the intestinal wall with small nodules appeared in the mucosa.

**Histopathology:**

**Acute stage:**

**Intestine:** The histopathological changes in the intestine were in the form of severe congestion of the blood vessels, necrosis and desqumation of the villar epithelium and prominent edema of the villus cores (Fig. 1). Also necrobiosis of the glandular epithelium could be noticed.

**Liver:** The hepatic cells showed degenerative changes of hydropic type. Prominent congestion fo the blood vessels with edema of the portal connective tissue and Disse spaces could be observed. The most characteristic changes were necrobiosis and necrosis of the bile ductal epithelium (Fig. 2).

**Kidneys:** The tubular epithelium showed a prominent degenerative changes of hydropic type with evidence of necrosis. The glomeruli were showing edema with dilatation of the Bawmans capsule and congestion fo the capillary tufts. Congestion of the interstitial blood vessels and edema of the interstitium could be noticed.

**Lungs:** The pulmonary changes were in the form of congestion of the blood vessels, fibrinoid degeneration of the vascular wall and perivascular and interlobular edema (Fig. 3). In the lobules, some areas showed atelectasis due to severe congestion of the septal capillaries and others were showing compensatory emphysema.

**Heart:** Myocardial degeneration with interstitial edema and congestion of the arterioles and capillaries could be observed.

**Spleen and Brain:** Only congestion of the blood vessels was observed.

**Chronic stage:**

**Intestine:** The histopathological changes which could be noticed in the intestine were chronic inflammatory processes. The Villar epithelium was showing a prominent proliferation, where it formed by more than 10 layers in some areas with hypermucin dystrophy (Fig. 4). Also the glandular epithelium showed proliferation and formed by more than one layer. The connective tissue cores of the villi were thickened due to lymphoid and heterophilic cell reaction.

In the mucosa, submucosa and sometime extend to the musculosa, a productive reactions in different stages could be noticed. Some areas showed histocytic and lymphoid cell reaction. In other locality, the productive reaction begin to be necrosed at the center with heavy cellular reaction surrounding these areas. Most of these reactions extended from the mucosa and submucosa to the musculosa (Fig. 5). Some of the productive reactions were forming typical chronic granuloma, where it consisted of complete central necrosis with connective tissue encapsulation (Fig. 6). Beside these local Productive reactions, a proliferation of the connective tissue extending from the submucosa to the mucosa inbetween the intestinal gland could be observed.

**Liver:** The hepatic cells were showing moderete degenerative changes of hydropic type. In some hepatic lobules, focal hepatic cell necrosis infiltrated by heterophils and lymphocytes could be noticed. In other areas, these necrosed hepatic cells with the cellular reaction completely encapsulated by connective tissue. The most prominent changes in the liver were in the portal triads. The bile ducts were showing epithelial hyperplasia with papillary projections. Also the proliferating epithelium showed necrobiosis and necrosis. In the portal triads, prominent connective tissue formation; periductal and perivascular; could be found (Fig. 7).

**Kidneys:** The most characteristic pathological changes in the kidneys were the prominetn mesengial cell proliferation in the glomeruli (Fig. 8). The tubular epithelium showed mild nephrosis.

**Lungs:** In the lunges, only prominent perivascular and interlobular cellular reaction of lymphoid type could be noticed.

**Heart:** The myocardium showed focal areas of necrosis and necrobiosis infilterated by lymphoid cells (Fig. 9).

**Spleen:** The splenic reactions manifested by lymphoid proliferation in both white and red bulbs with increasing number of plasma cells (Fig. 10).

## *DISCUSSION*

The clinical and gross pathological findings observed in the present study were typically as those described previously by AWAAD (1972); PIERCY and WEST (1976) and ADLER and DAMASSA (1977) in both naturally and experimentally infected birds. As the symptoms appeared three days after inoculation, so we suggested that, this is the incubation period of the disease. Gradually disappearance of the symptoms and mortality are due to localization of the organism and development of bird resistance.

The histopathological changes in dead birds were mostly related to circulatory disturbances, induced by the organism or its toxins. These circulatory disturbances were severe congestion of the blood vessels of all organs and interstitial edema in the intestine, liver, lung, kidney and heart. The necrobiotic and necrotic changes of the intestine, liver and kidney, may be attributed to the direct action of the organism and its toxins. Also it may be due to central circulatory disturbance resulted from myocardial degeneration induced by the causative agents or its toxins.

The available literatures in avian colisepticaemia were mainly concerned with the description of clinical signs and gross pathological changes.

Sufficient histopathological data is still lacking in comparison to mammals, which was studied by several investigators. Our results of acute coli septicaemia in birds were in agreement with those described in mammals by many workers in natural and experimental coli enterotoxaemia (KURTZ, BERGLAND and BARNES, 1969; MOON 1970; CLOOSTON, NIELSEN and SMITH, 1974; BUROW, 1975 and DROMMER, 1976).

The fibrinoid degeneration of the vascular wall of the blood vessels which was noticed in the lungs was described by KURTZ et al. (1969) and CLUGSTON, et al. (1974) in mammals induced by coli infection or its toxins.

Granulomatous reactions, connective tissue proliferation and epithelial hyperplasia of the villi and bile ducts were observed in the intestine and liver of chronic stage. The coligranuloma described in our results in intestine and liver was as that observed by HJARRE and WRAMBY (1945) and HAMILTON and CONRAD (1958). The increase of connective tissue in the mucosa and submucosa of the intestine and hepatic portal triads was not described previously in coli infection of birds, but these findings were described by BUROW (1975), DROMMER (1979) and NAFADY (1981) in protracted shock induced by coli toxin in swine and rats. The authers attributed the connective tissue proliferation to stimulating factors found in edema induced in acute shock. ANDERSSON, MELCHERS, GALANES and LUDERTZ (1973) and VAHERI, RUESLAHTI, SERVAS and NURMINE (1973) found the endotoxin of E.coli having a mitogenic effect on the fibroblast and lymphoid cells. In our results, interstitial edema in intestine and liver in a cute stage was followed by increases of the connective tissue in the interstitium. We suggested that, the fibroplasia was induced by the two factors together, either the stimulating factors of edema or direct action of the coli toxin.

The epithelial proliferation of the intestinal villi and hepatic bile ducts recorded by the authers was not described previously in coli infection of birds. We suggested its induction to the reparative changes of the epithelial necrosis which was noticed in acute stage.

We attributed the lymphoid reactions in the spleen, intestine and lung to immune response against the coli infection.

Our results concerning mesangial cell proliferation of the glomeruli in the kidney were closly resembling those described by DROMMER, VELTMAN and SCHULZ (1980) in swine suffering from coli-toxin shock.

Myocardial degeneration and lymphoid cell infilteration described by GROSS (1966) in birds infected by E.coli, were similar to those found in the present study.

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Fig. (5)

Fig. (6)

Fig. (7)

Fig. (8)

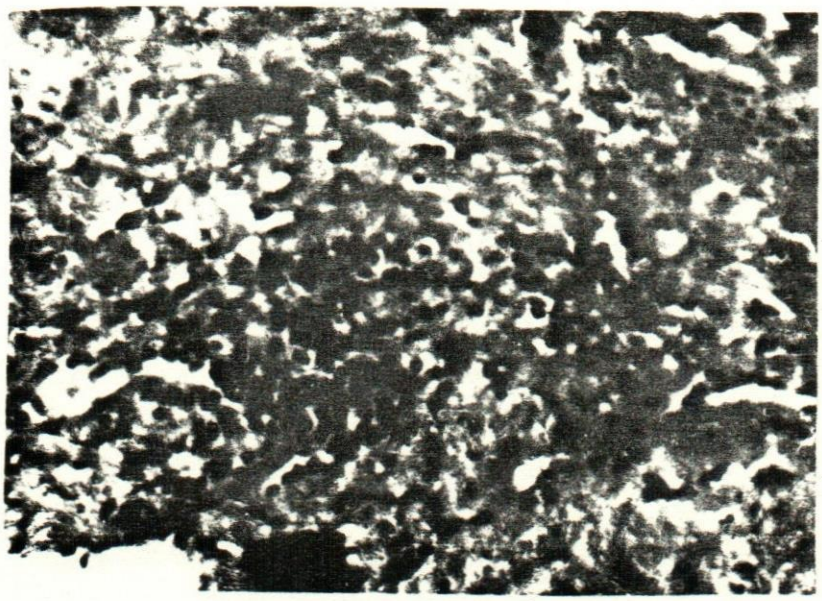

Fig. (9)

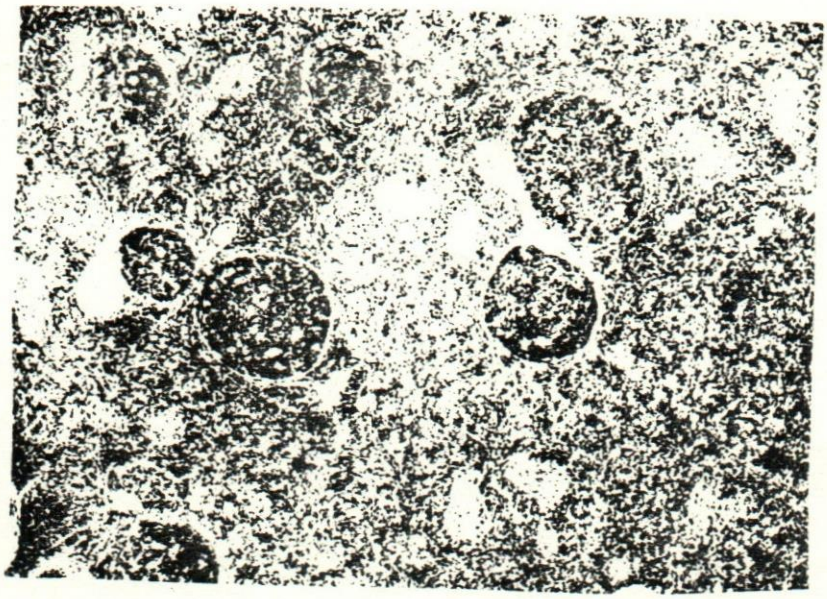

Fig. (10)