PATHOLOGICAL STUDIES ON COLIBACILLOISIS IN CHICKEN

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Abstract

Twenty chicks of one day age of Cubb breed,obtained from El-Kahera Company for Poultry, fed a balanced ration. The chicks will be classified into two groups. Group(A)of 20 chicks infected with E. coli strain O78 in a dose of 108 cfu S/C. at the 5th day of age (Goten, 1976).. Group (2) contain 10 chicks, used as control, 5 chicks were sacrificed after 15 and 21 day from infection. The specimens were collected from lungs, air sacs, heart, liver and intestine and inoculation on MacConkey's media and incubation at 37°C for24-48 hours. Positive cases gave bright pink colonies Specimens from Live, kidney, lungs, air sacs, heart, spleen, joints and intestine were taken for histopathological examination and fixed in 10% neutral buffer formalin, paraffin sections of 5 micron thickness were prepared, stained with H&E stain then examined microscopically. The infected birds showed depression,weight loss, diarrhea and mortality rate 65% within the first 7 days post infection(PI).poor growth and labored rapid breathing with gasping,rales andcoughing,15 days PI. Microscopically, Liver,showed severe congestion of the hepatic blood vessels and sinusoids, with extensive hemorrhages were seen 15 days PI . The hepatocytes suffered vacuolar degeneration , coagulative necroses, infiltrated with lymphocytes and heterophils . Perihepatitis and thickened hepatic capsule by serofibrinous exudates and leukocytes were recorded 21 dayes PI . . The lesions of heart were severe and charecterized by congested capillaries, extensive hemorrhages and heterophils infiltrations on the coronary fat.15 &21days PI .Thick pericardium with focal caseous necrosis beside granulation tissue and heterophils infiltration were seen .The myocardium focally replaced by macrophages, few heterophils and lymphocytes . Organized thrombus was recorded beside perivascular aggregation of macrophages and lymphocytes . Intestine 15 dayes PI showed necrotic mucosa with a cap of pseudomembrane . The submucosal infiltrated with macrophages, lymphocytes, plasma cells and heterophils. 21 days PI.Fig (11) . The mucosa suffered necrosis with denuded villi from the enterocytes . Thickening of the bronchial wall with congested capillaries, edema, extensive hemorrhage and inflammatory cells were seen in the pulmonary tissues 15 &21 daysPI with peribronchial (tertiary) aggregation of histiocytes, lymphocytes and few heterophils . Peri vascular edema and lymphocytic cuffing wer also seen . Kidneys showed congested blood vessels and cappillaries with extensive hemorrhages among the renal tubules ,15days PI .Varriable density of eosinophilic and basophilic radiating crystals of urates was detected inside the renal tubules 21days PI .Aggregations of round cells among the renal tubules was observed .

INTRODUCTION

The production of meat type poultry has greatly expanded over the post several decades The commercial broiler industry has evolved from backyard flocks into an ingenious mass feed system. Growth and yield birds have been enhanced with the help of highly specialized diets and control of the systemic diseases particularly of bacterial

origin through the use of broad spectrum antibiotics. Colibacilloss is a serious disease causing a great losses in poultry. Also, it refers to any localized infection or systemic infection causes entirely or partly by E. coli It includes(coliseptecemia, coligranuloma chronic respiratory disease (CRD) peritonitis, arthritis and synovitis, (Gross, 1991).

This work was planned to study the experimental infections by *E. coli* in chickens employing the clinical signs, and pathology with microbial identification.

MATERIALS AND METHODS

Thirty chicks of one day age of Cubb breed. The chicks were obtained from El-Kahera Company for Poultry. The chicks were fed a balanced ration and classified into 2 groups. Group A contain 20 chicks and infected with E. coli strain O78 in a dose of 108 cfu intraperitoneal at the 5th day of age (Goten, 1976). Group B contain 10 chicks, used as control, 5 chicks were sacrificed after 15 and 21 day from infection

II. Methods

The specimens were collected from lungs, air sacs, heart, liver and intestine and inoculation on MacConkey's media and incubated at 37°C for24-48 hours. Positive cases gave bright pink colonie (Gross, 1991).

Specimens from lungs, air sacs, heart, liver, kidneys, and intestine were taken for histopathological examination and fixed in 10% neutral buffer formalin, paraffin sections of 5 micro thickness were prepared, stained with H&E stain then examined microscopically (Bancroft and Stevens, 1996.

RESULTS AND DISCUSSION

The infected birds showed depression,weight loss, diarrhea and mortality rate 65% within the first 7 days post infection(PI).poor growth and labored rapid breathing with gasping,rales and coughing,15 days PI.Such findings were similarly to those discribed by Ramadan,(1996) and Manakarious,(1999). The mortality may be due to colisepticemia with production of severe lesions in the vital organs (EL-Attar, 1985). Macroscopically,the dead&sacrifieced birds showed septicemic lesions-scores represented by generalized congesion of the internal organs and hemorrhages on the coronary and abdominal fat,epicardium,intestinal mucosa and skeletal muscles.Grayish or hemorrhagic necrotic areas on the surfaces of liver ,kidney and lungs beside acute catarrhal and necrotic enteritis.Thickening of the pericardial sac

was observed with adherent caseous daposits.Geayish –white nodules of 3-5 mm in diameter were seen in the liver.epicardium and lungs. The air sacs were thick ,dull,opaque with focal white caseous material. The previous lesions may be as a result of colisepticemia and endotoxins of the microbebeside localization of the bacteria in the pericardium and lungs Similar findings were recorded by Shihata et al. ,(1983) , Jordan,(1990) and Mohamed and fouad (2004). Microscopically, Liver,showed severe congestion of the hepatic blood vessels and sinusoids, with extensive hemorrhages were seen 15 days PI Figs.(1&2.) The hepatocytes suffered vacuolar degeneration , coagulative necroses, infiltrated with lymphocytes and heterophils Figs.(3&4). Perihepatitis and thickened hepatic capsule by serofibrinous exudates and leukocytes were recorded 21 dayes PI Fig.(5). Capsular and subcapsular infiltration with numerous macrophages, lymphocytes and fibroblasts Fig.6.Similar findings were recorded by Ramadan 1996 and Mankarious (1999). The lesions of heart were severe and charecterized by congested capillaries, extensive hemorrhages and heterophils infiltrations on the coronary fat.15 &21days PI Fig.(7). Thick pericardium with focal caseous necrosis beside granulation tissue and heterophils infiltration were seen Fig(8).The myocardium focally replaced by macrophages, few heterophils and lymphocytes . Organized thrombus was recorded beside perivascular aggregation of macrophages and lymphocytes Fig.(9) . Intestine 15 dayes PI showed necrotic mucosa with a cap of pseudomembrane Fig.(10) . The submucosal infiltrated with macrophages, lymphocytes, plasma cells and heterophils. 21 days PI,Fig (11) . The mucosa suffered necrosis with denuded villi from the enterocytes Fig (12). Thickening of the bronchial wall with congested capillaries, edema, extensive hemorrhage and inflammatory cells were seen in the pulmonary tissues 15 &21 days PI with peribronchial (tertiary) aggregation of histiocytes, lymphocytes and few heterophils figs.(13&14). Peri vascular edema and lymphocytic cuffing wer also seen . Kidney showed congested blood vessels and cappillaries with extensive hemorrhages among the renal tubules ,15days PI fig.(15) Variable density of eosinophilic and basophilic radiating crystals of urates was detected inside the renal tubules 21days PI fig(16). Aggregations of round cells among the renal tubules was observed . (Abou EL-Nil 1997) found renal damages in chicks infected with E.coli due to septicemia or bacterial toxins.

Generally,the histopathological changes in dead birds were septicemic lesions and mostly related to circulatory disturbances caused by the organism or its endotoxins .These circulatory disturbances represented by congesion ,hemorrhages ,thrombosis of blood vessels ,together with degenerative changes and necrosis in the liver and kidneys. (Nafady et al 1983 and Harrison and Harrison ,1986).

Fig. 1 : Liver,15days PI, showing severe congestion of the hepatic blood vessels and sinusoids (arrows), HE x 150.

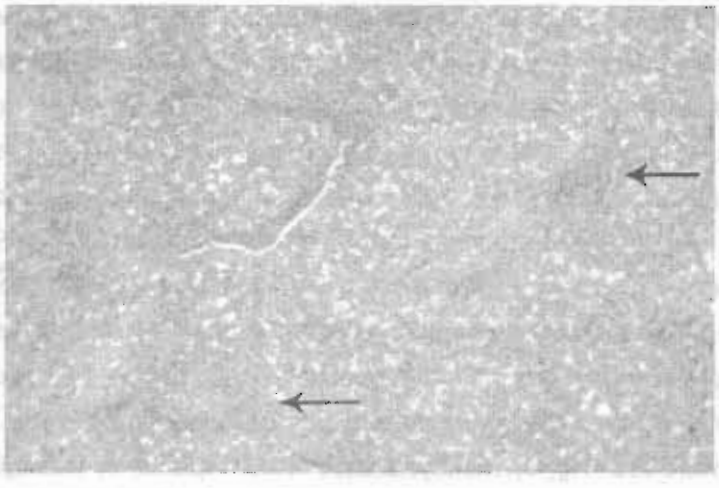


Fig.2 : Liver 15daysPI, showing,extensive hemorrhages among the hepatic cells (arrow) . HE x 1200.

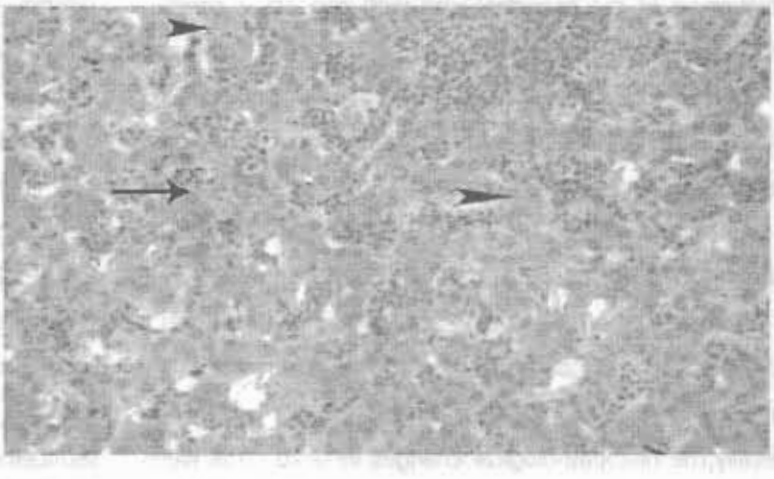


Fig.3: Liver,15days PI,the hepatocytes suffered vacuolar degeneration (arrowhead) HEx1200.

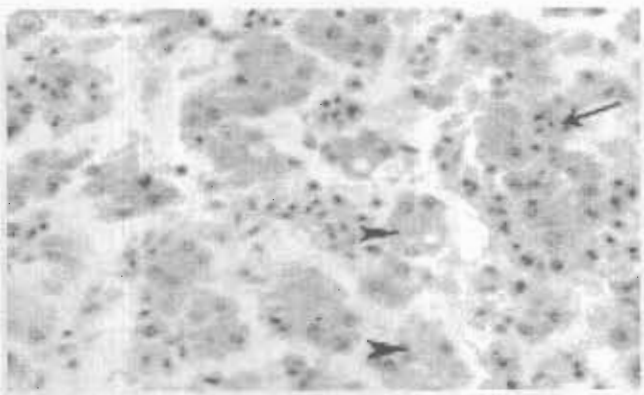


Fig.4: Liver,15days PI, coagulative necroses, infiltrated with lymphocytes and heterophils.(arrow). HEx1200.

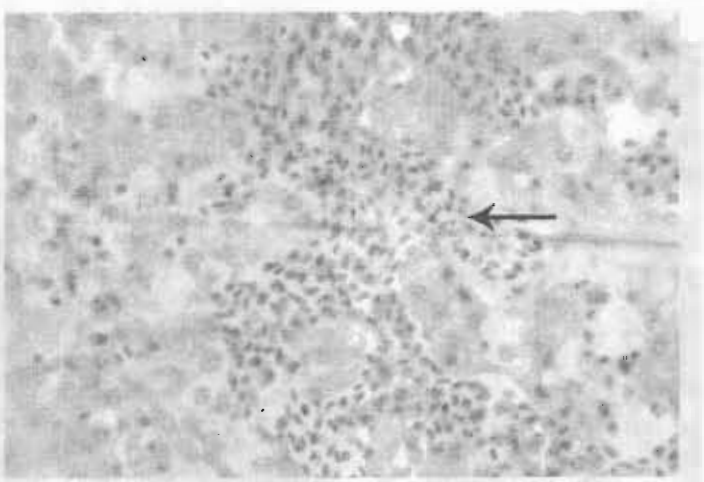


Fig.5: Liver 21 days,PI ,showing, perihepatitis and thickened hepatic capsule by serofibrinous exudates and leukocytes(arrow) . HEx150

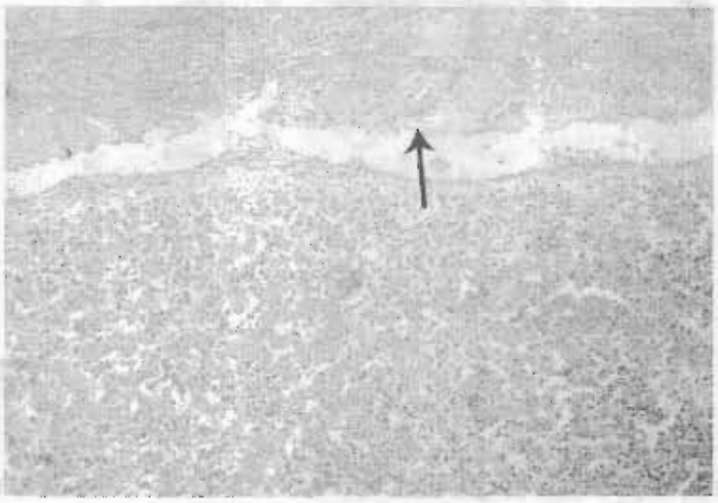


Fig.6: Liver,21daysPI, showing,capsular and subcapsular infiltration with numerous macrophages, lymphocytes and fibroblasts (arrow) HE x 1200.

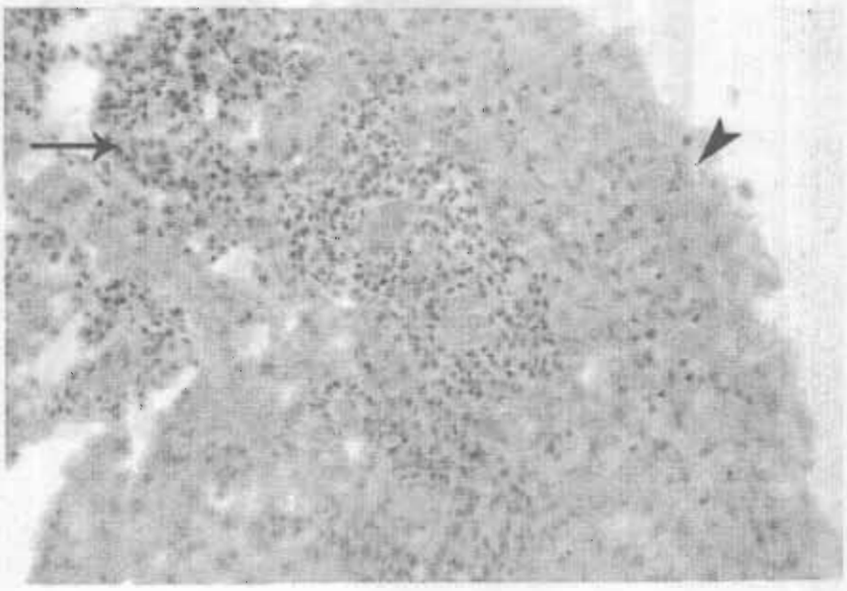


Fig.7: heart 21days PI showing congested capillaries, extensive hemorrhages and heterophils infiltrations on the coronary fat . HE x1200

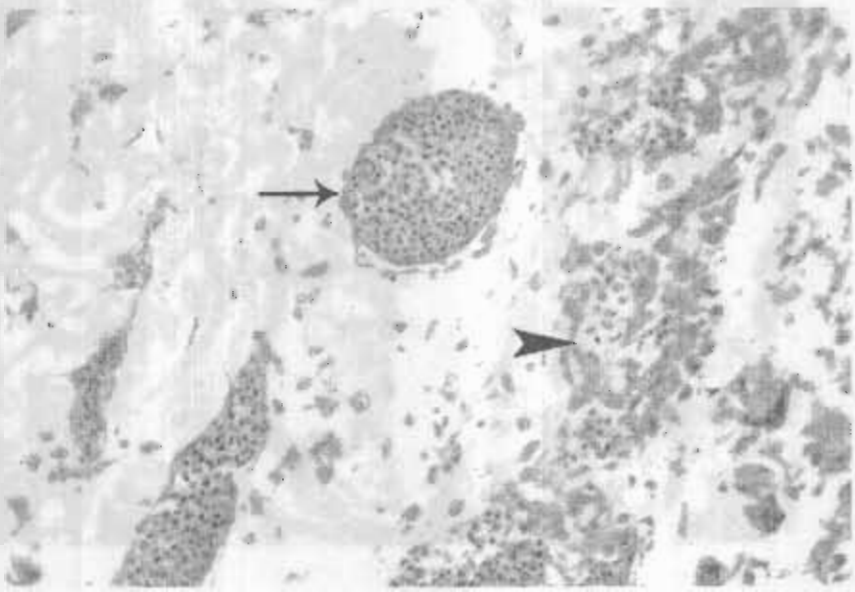


Fig.8: Heart 21daysPI showing,thick pericardium with focal caseous necrosis beside granulation tissue and heterophils infiltration . HEx150.

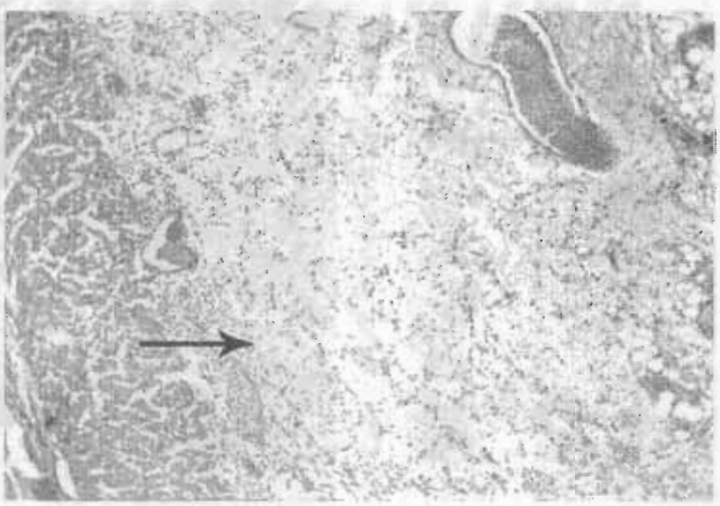


Fig.9: Heart 15 days PI, showing organized thrombus (arrow). HE x 1200.

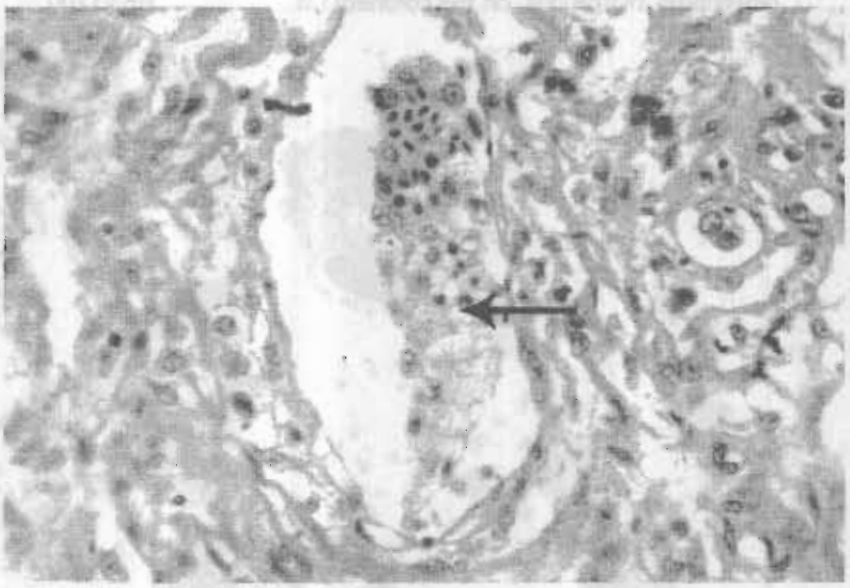


Fig.10: Intestine 15 days PI showing,necrotic mucosa with a cap of pseudomembrane. H&Ex300.

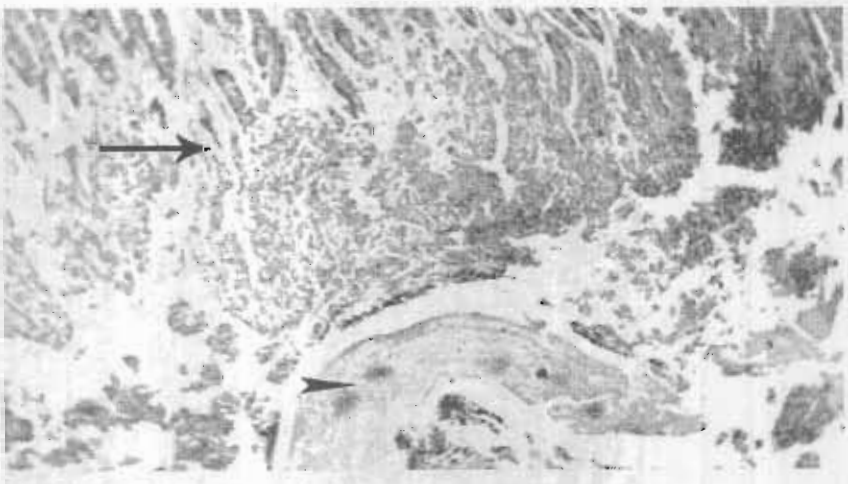


Fig 11: Intestine. 21 days PI showing submucosal infiltration with macrophages, lymphocytes, plasma cells and heterophils. HEx1200.

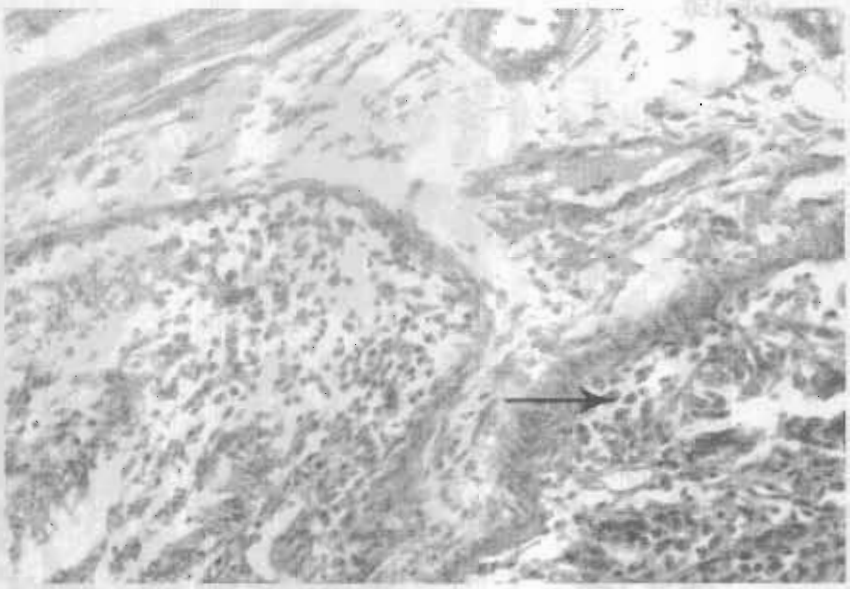


Fig.12: Intestine 21 days PI, showing ,the submucosa with macrophages, lymphocytes, plasma cells and heterophils infiltration (arrow). HE x 1200

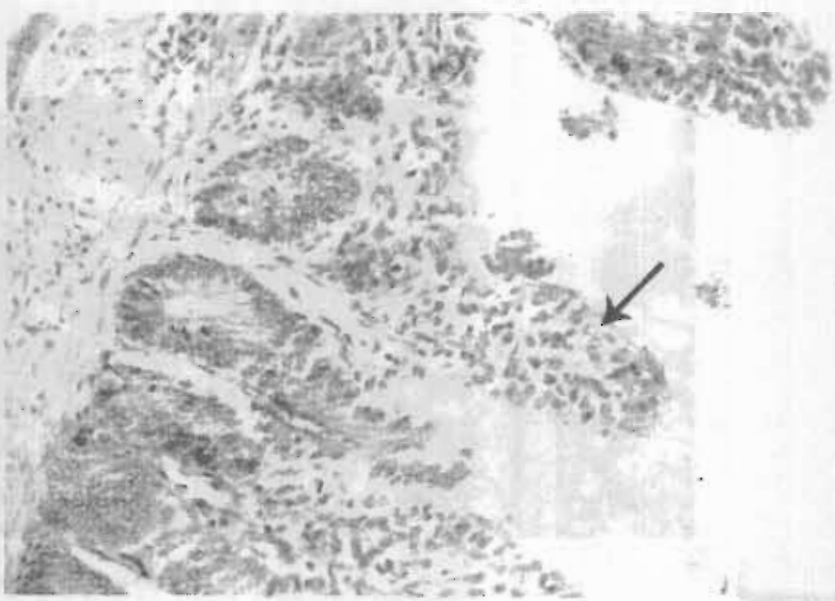


Fig.13 :Lung.15 days PI,showing thickening of the bronchial wall with congested capillaries, edema, extensive hemorrhage and inflammatory cells(arrow) HEx150.

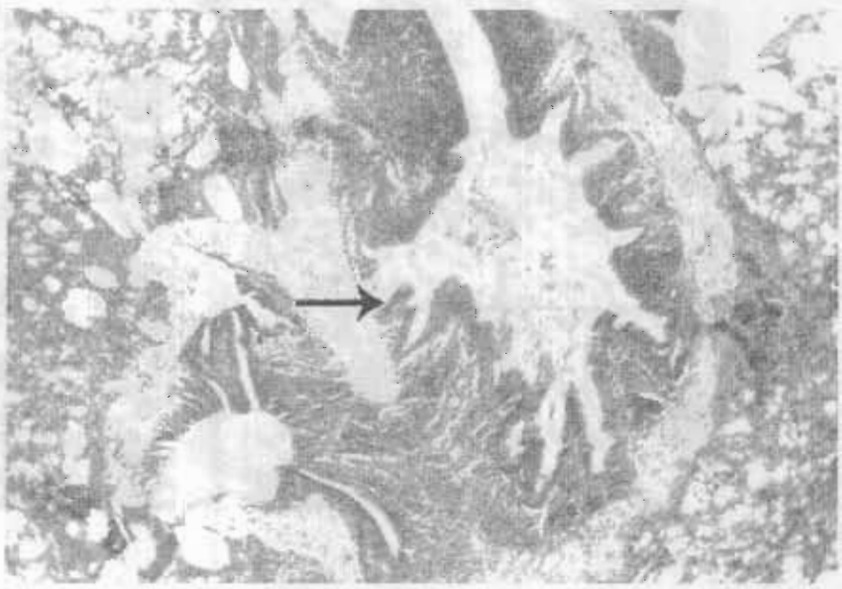


Fig.14 :Lung.15 daysPI, peribronchial (tertiary) aggregation of histiocytes, lymphocytes and few heterophils(arrow). HEx300

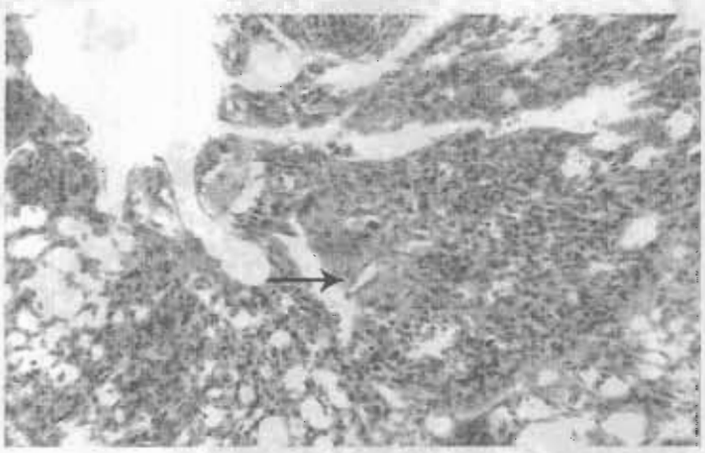


Fig.15: Kidney,15 dayS PI, showing, congested blood vessels and cappillaries with extensive hemorrhages among the renal tubules(arrow) HEx150.

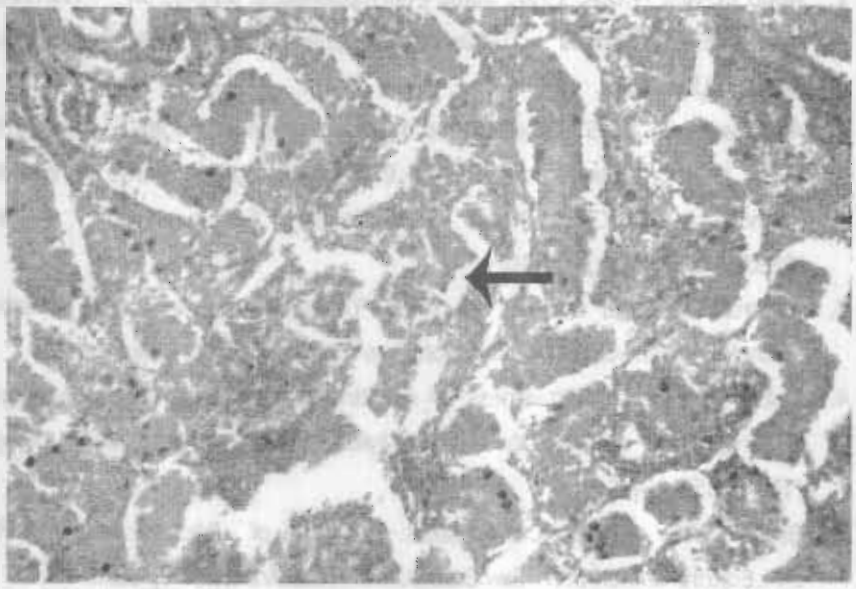
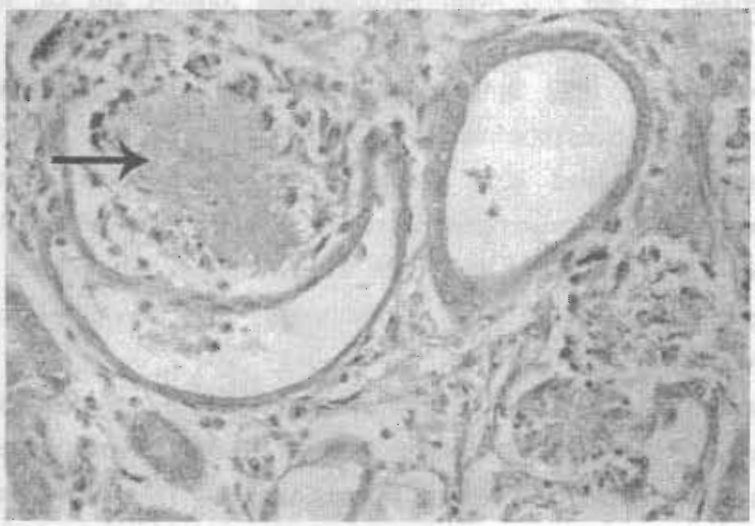


Fig.16: Kidney,21 days PI, showing,basophilic radiating crystals of urates in the renal tubules(arrow) HEx1200.



REFERENCES

1. Abou EL-Nile M.O.A. 1997. Some Pharmacological studies on Norfloxacin in experimental animals . Thesis M.V.Sc.(pharmacology) Faculty of Vet.Med., Zagazig University.
2. AL-Attar E.R. 1985. Experimental histopathological studies on the effect of some entero-pathogenic types of Escherichia coli in some experimetal animals. M.V.SC., Thesis (pathology), Faculty of Veterinary Meedicine,Zagazig University
3. Bancroft J,D and Stevens A. 1996. Theory and Practice of Histological Technice.4thed.,Churchill,Livigston,NewYork,London,SanFrancisco.
4. Goten E. 1976. Effectiveness of spectinomycin against avian mycoplasma cultures and resistance with tylosin. Tijdschrift voor Diergen,97 (18):1185-1188.
5. Gross W.B. 1991. Colibacillosis disease of Poultary.9th Ed.,PP.138-144.Editors:Calnek B.W. Iow State,Univ. Press.Ames Iowa, USA.
6. Harrison G.H. and Harrison L.R. 1986. Clinical Avian Medicine and Surgery . W.B. Saunders Company ,Philadelphia ,London,Toronto.
7. Jordan F.T.W. 1990. cited in Handbook of poultary diseases ,Enterobacteriacaea and Avian Mycoplasmosis ,Chaptrt 3rd Ed. English Language Book Sociaty , Bailliere.
8. Mankarious E.E. 1999. Some pharmacological studies on Ciprofloxacin in chicken .Ph.D, Thesis (pharmacology) Fac. Of Vet. Med. Zagazig university.
9. Mohamed and Fouad. 2004. Studies on the effect of E.coli on ocular, subcutaneous and ovarian tissues in broilert chickens . Assiut Vet. Med.J., 50.
10. Nafady A., Ibrahim A.and Shehata M. 1983. Pathological Picture of E.coli serogroup o124:k279B120 in experimentally infected chicks .Assiut Vet.Med.J.,14(27)..
11. Ramadan O.E. 1996. Some pharmacological studies onDanofloxacin in chicken. Ph. D, Thesis (pharmacology) Fac. Of Vet. Med. Zagazig university.
12. Shihata A.B., el-Taher A. , NasserA.A. and SalamaA.A. 1983. The intestinal flora of Egyptian doses (Colama oenas) with special references to E.coli in Sharkia province. J.Zag.VetMed., VII:63-72.

دراسات باثولوجيه على عدوى الميكروب القولونى فى الدجاج

ابراهيم السيد محمد احمد

معهد بحوث صحه الحيوان - الزقازيق

تم اجراء هذا البحث على عدد 30 كتكوت عمر يوم من شركه القاهره للدواجن قسمت الى مجموعتين الاولى 20كتكوت تم حقنها داخل التجويف البريتونى بميكروب القولونى o78 والمعزول من دجاج ظهر عليه اعراض تنفسيه فى مزارع التسمين ويتم الذبح بعد 5و 21 يوم بعد الحقن والمجموعه الثانيه 10كتاكيت تركت كضابط للتجربه كانت نسبه النفوق 65% وقد اظهر الفحص العينى عند اجراء الصفه التشريحيه وجود تغير فى لون الكبد مع مناطق متنكرزه اما القلب فقد بين عتامه مع زياده فى سمك الغشاء التاموري والامعاء بها احتقام مع محتويات مائيه والرئتين كان بها مناطق التهابيه اما الكليتين فبها احتقان مع زياده فى الحجم اما الفحص الميكروسكوبىفقد بين احتقان ونزف وتغيرات استحاليه واوديما وتنكرز مع احتقان وتجمع سائل مصلى فيبرينى فى غشاء القلبمع ارتشاح للخلايا العملاقه وجلطات فى القلب اما الامعاء فقد بينت تغيرات التهابيه كذلك شوهد احتقان ونزف والتهاب فى الشعب بالرئتين اما الكليتين فقد اظهرت احتقان ونزف وتنكرز وقد اظهرت النتائج خطوره الميكروب القولونى على الدواجن بزياده نسبه النفوق والتغيرات الباثولوجيه