Journal of Pharmacognosy and Phytochemistry
Available online at www.phytojournal.com

E-ISSN: 2278-4136
P-ISSN: 2349-8234
JPP 2018; 7(5): 798-801
Received: 21-07-2018
Accepted: 22-08-2018

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Pathomorphological changes in broiler chickens due to spontaneous *E. coli* infection

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Abstract
A study was carried out round the year from May 2015 to April 2016 to establish the pathomorphological changes in the broiler chickens due to spontaneous *E. coli* infection in Faizabad and Sultanpur districts of Uttar Pradesh. For this study a total number of 178 naturally dead broiler chickens from different private farms of Faizabad district and 151 naturally dead broiler chickens from Sultanpur district were collected throughout the year. The birds were of different age group and of both sexes. After post mortem examination, the grossly suspected samples were kept for further bacteriological study. Post mortem examination was conducted for all birds (178+151) and different internal organ were examined for the presence of gross lesions. The gross change of liver was found as enlarged, congested and covered with thick yellow/white serofibrinous covering. Haemorrhages, congestion and edematous swelling were noted in intestine. Heart was also covered with thick yellow/white serofibrinous covering. Among tested 178 and 151 samples from Faizabad and Sultanpur districts, 95 (53.37%) and 44 (29.13%) respectively were found to be suggestive of *E.coli* infection in bacteriological study. In histopathological study, the liver of the infected birds showed extensive haemorrahage, necrosis of hepatic cells and thickening of liver capsule, cellular infiltration and fatty changes. In intestine, there were congestion, severe necrosis of villi and intestinal gland, cellular infiltration and oedema of submucosa. Heart showed congestion, severe cellular infiltration, presence of thrombus and thick pericardium. Lungs showed congestion, thrombus and emphysematous changes in few cases. Spleen were congested and necrosed with thick splenic capsule.

Keywords: Broiler, *E. coli*, pathomorphology

Introduction
Avian colibacillosis is one of the most common infectious diseases seen in poultry of all age group and usually found in young chick of up-to three weeks of age. It occurs in all poultry as well as in other birds and mammals. Colibacillosis is an important cause of mortality in poultry of younger age than older one (Kabir, 2010) [9]. Economically the disease is very important as it causes heavy mortality producing heavy economic loss to the poultry farmers. The bacterium has over a hundred serogroups with the most common and pathogenic ones being O8, O1, O2, O15 and O55 (Kabir, 2010) [9]. Pathogenic *E. coli* strains have been divided into intestinal pathogenic *E. coli* and extra-intestinal pathogenic *E. coli* (ExPEC) depending on the location of the infection. Avian pathogenic *Escherichia coli* (APEC) strains belong to the ExPEC group is a major pathogen responsible for morbidity and mortality in chickens (Ashraf *et al.*, 2014) [1]. Morbidity varies but mortality ranges from 5-20%. Avain Pathogenic *E. coil* (APEC) has been incriminated in a lot of cases *in* both broilers and layers. It causes embryo mortality and omphalitis in chicks. Lesions observed are mainly polyserositis with deposition of fibrin in the air sacs, pericardium and liver (Yousseff *et al.*, 2008; Kabir, 2010) [13, 9]. It is usually followed by a systemic infection with characteristic fibrinous lesions (airsacculitis, perihepatitis and pericarditis) and fatal septicemia (Sharada *et al*., 2010) [11]. The pathogenicity of *E. coli* is generally enhanced or initiated by predisposing factors, such as mycoplasma infections, viral infections, environmental factors and immune-suppressive diseases (Gomes *et al*., 2005) [8]. Zoonotically, the disease is very important as poultry meat is the richest source of protein to human population of the world (Ewers *et al*., 2003) [7]. Amongst poultry diseases transmissible to human being, avian colibacillosis is of great concern. Keeping in view the immense importance of this disease in broiler industry, the present study was undertaken to investigate pathomorphological changes due to spontaneous *E. coli* infection in broiler chickens in Faizabad and Sultanpur districts of Eastern Uttar Pradesh.

Materials and methods
For the present study, a total number of 178 naturally dead broiler chickens from different private farms of Faizabad district and 151 naturally dead broiler chickens from Sultanpur

district were collected within 6 hours of death throughout the year (from May 2015 to April 2016). The birds were of different age group and of both sexes. After post mortem examination, all the gross lesions were recorded. For bacteriological study, materials from grossly suspected birds for *E. coli* infection were collected. The pieces of liver, lungs, heart, intestine, and spleen were aseptically collected from the dead birds showing gross pathological lesions of suspected *E. coli* infection i.e. yellow/white sero-fibrinous covering over liver, heart, and peritoneum, congestion of lung and heart, intestinal haemorrhages and necrotic foci on liver. They were kept in sterile vials separately and brought to the Veterinary Microbiology Laboratory under ice coverage for further study. The method described by Cruickshank *et al*. (1975) [4] was used for the isolation of *E. coli* and the isolates were identified on the basis of morphology, motility, and colony characteristics and bio-chemicals properties as per the method of Edwards and Ewing (1972) [6]. From the same dead birds showing gross pathological lesions of *E. coli* infection, pieces of liver, lung, heart, intestine and spleen were also collected and immediately fixed in 10% buffer formal saline solution. Tissue pieces of different organs preserved in 10% formalin solution were processed for paraffin sections (3-5 µ thick). Sections were stained with Mayer's haematoxylin and eosin for histopathological examinations (Bancroft and Stevens, 1980) [2].

Results and discussion

Gross Pathological Changes

Gross pathological lesions observed during necropsy in birds of different districts in the present study are summarized in Table 1 and 2. The gross changes of liver were found as enlarged, congested and covered with thick yellow/white sero-fibrinous covering due to spontaneous *E. coli* infection in broiler chickens. The serofibrinous membrane covered the liver either partially or completely that differs with degree and severity of infection. In intestine haemorrhages, congestion and oedematous swelling were noted. Heart was also covered with thick yellow/white sero-fibrinous covering (Fig. 1). In severe *E. coli* infection the serofibrinous membrane covered all the visceral organs continuously (Fig. 2). The gross lesions observed in the present study corroborated with the findings of Tonu *et al.* (2011) [12], Daud *et al*. (2014) [15] and Parwez *et al*. (2015) [10].

Table 1: Summary of gross pathological lesion observed in necropsy of birds (n=178) in Faizabad district.

Post mortem finding	No. (%) in population
Fibrinous pericarditis	89(50%)
Fibrinous perihepatitis	85 (47.75%)
Airsacculitis	82 (46.07)
Fibrinous peritonitis	78 (43.82%)
Pale breast muscle	10 (5.6%)
Liver congested	7 (3.93%)
Gizzard erosion	1 (0.56%)
Hydropericardium	1 (0.56%)

Table 2. Summary of gross pathological lesions observed in necropsy of birds (n=151) in Sultanpur district

Post mortem finding	No. (%) in population
Fibrinous pericarditis	75(49.66%)
Fibrinous perihepatitis	70 (46.35%)
Airsacculitis	68 (45.03)
Fibrinous peritonitis	61 (40.39%)
Pale breast muscle	7 (4.63%)
Liver congested	5 (3.3%)
Gizzard erosion	0
Hydropericardium	0

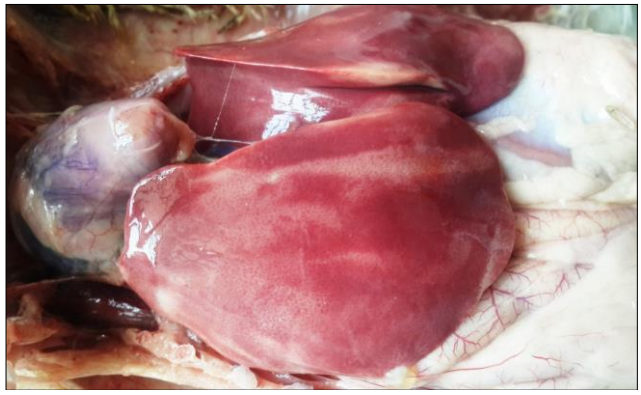

Fig 1: Colibacillosis affected bird showing pericarditis, perihepatitis, and congestion of liver, heart, and intestine.

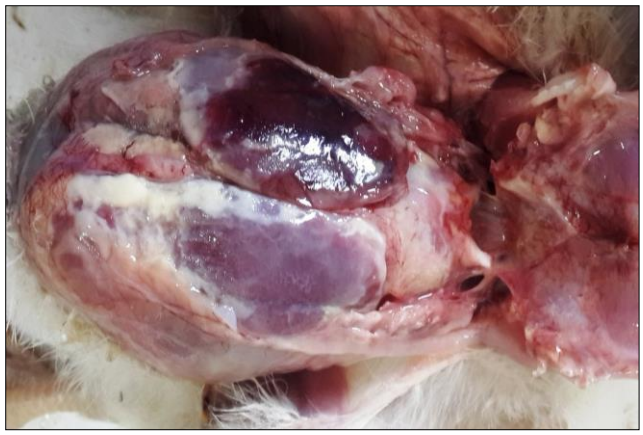

Fig 2: Colibacillosis affected bird showing thick white serofibrinous membrane that covers the visceral organs continuously.

Histopathological Changes

In the present study the histopathological changes due to *E. coli* infection in broiler chicken were observed mainly in liver intestine, lung, heart and spleen. Under microscopic observation, there was severe congestion of the blood vessels in the liver (Fig. 3), extensive haemorrhages and necrosis of hepatic cells (Fig. 4) which differs in degree and severity of infection in different birds. There was thickening of liver capsule along with infiltration of inflammatory cells and extensive fatty changes (Fig. 5). Similar findings in the liver were also reported by Chandra *et al*. (2008) [3]. The intestine showed severe necrosis of villi that were completely filled with inflammatory cells (Fig. 6) which differs in degree and severity of infection in different birds. There was also severe glandular necrosis and congestion. Large numbers of goblet cells were found in the mucosal layer in some of the sections of intestine indicating catarrhal inflammation in mild infection of *E. coli*. The submucosa was markedly oedematous. The above intestinal changes simulated with the reports of Tonu *et al*. (2011) [12]. Section of the heart showed congestion and severe cellular infiltration (Fig.7). In few cases there was presence of thrombus along with proliferation of vascular wall. Thick pericardium along with cellular infiltration was most common findings. Haemorrahages were also found in some sections between the cardiac muscle fibres. The above cardiac changes were corroborated with the findings of Daud *et al.* (2014) [15]. Histopathological observation of lungs showed congestion (Fig. 8). A few birds showed emphysematous changes in the lung (Fig. 8). Presence of thrombus was also noted in few birds infected with colibacillosis. These findings were also reported by Tonu *et al.* (2011) [12]. Microscopic examination of the tissue

section of the spleen revealed severe congestion and necrosis of lymphoid follicles (Fig. 9 & 10). There was thickening of spleenic capsule along with infiltration of numerous inflammatory cells. These findings were in accordance with Chandra *et al*. (2008) [3].

The present investigation throws light on the incidence and pathomorphological changes due to *E. coli* infection in broiler chicken. Many stress factor that lowered body defence mechanism, bad sanitary practice, poor ventilation, high density of chicken etc. can induce *E. coli* infection as *E. coli* is a common inhabitant of intestinal tract at a concentration of 10^6/gm of faeces. So this infection can be prevented by regular preventive treatment using antimicrobial agents along with improved sanitation, hygiene and better husbandry practice.

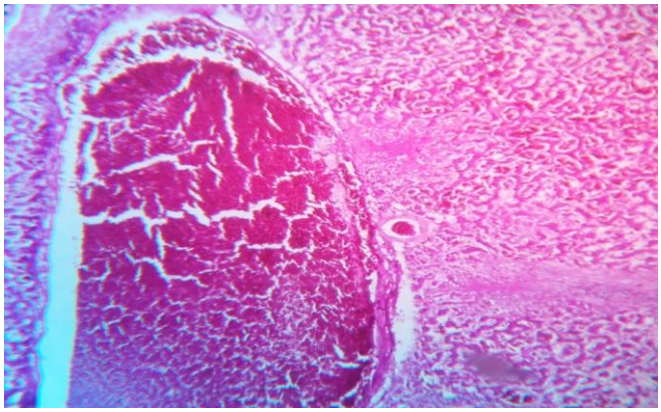

Fig 3: Section of liver showing severe vascular congestion and necrosis of hepatocytes (H & E X 200).

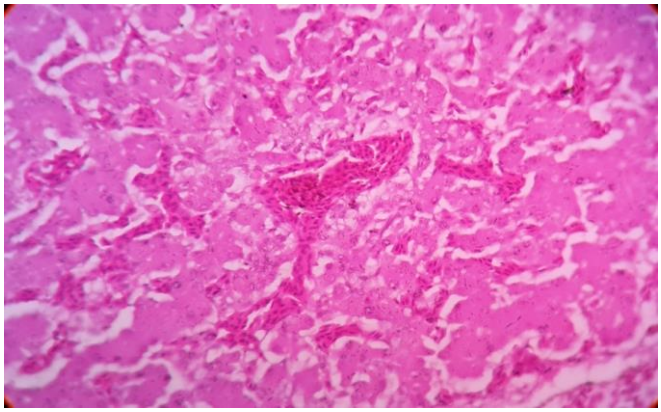

Fig 4: Section of liver showing haemorrhages and coagulative necrosis of hepatocytes (H & E X 400).

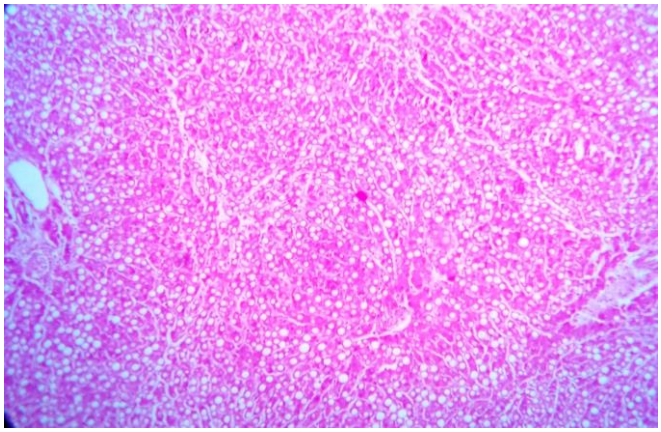

Fig 5: Section of liver showing extensive fatty changes (H & E X 200).

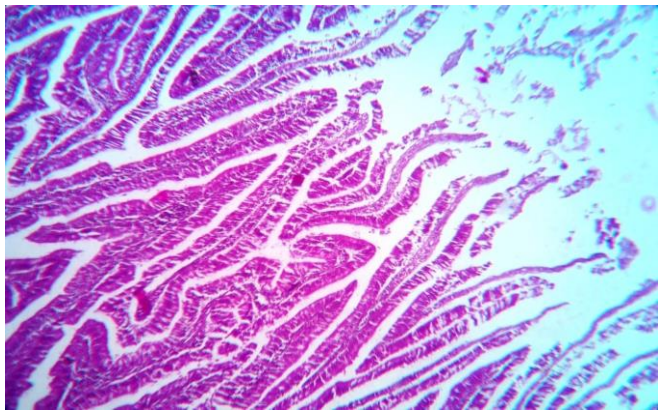

Fig 6: Intestine showing severe necrosis of villi and infiltration of inflammatory cells (H & E X 400).

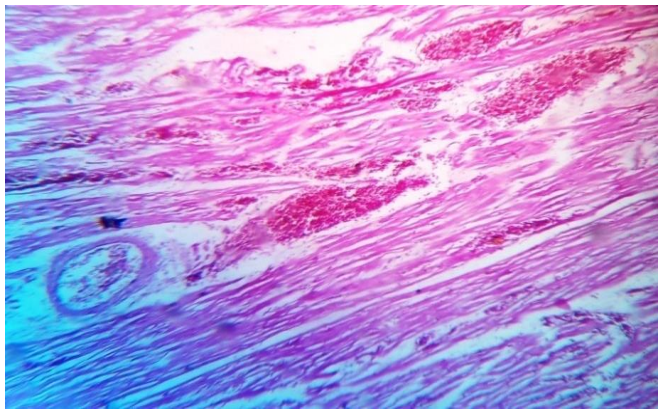

Fig 7: Section of heart showing congestion and severe cellular infiltration (H & E X 200).

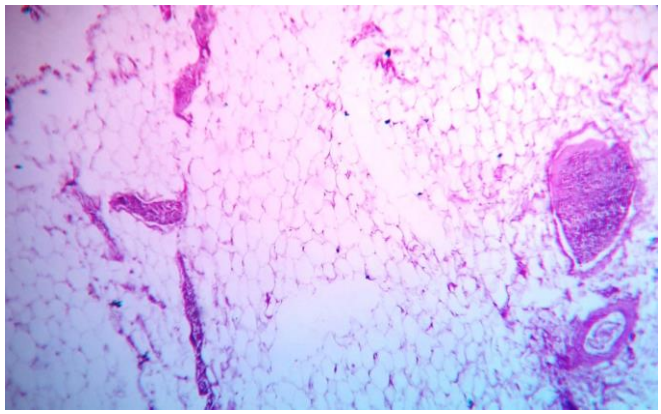

Fig 8: Section of lung showing congestion and emphysema (H & E X 200).

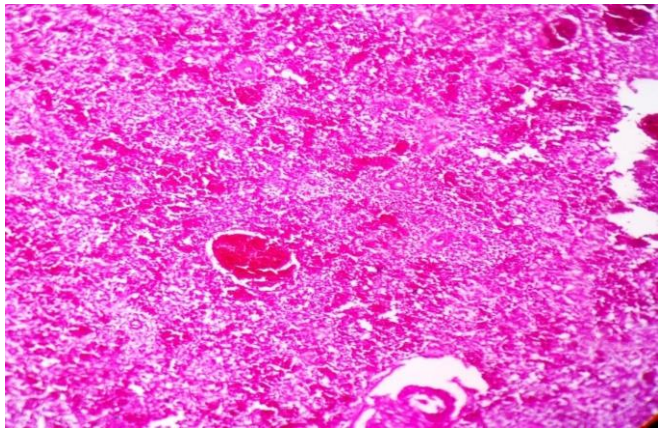

Fig 9: Section of spleen showing severe congestion and necrosis of lymphoid follicles (H & E X 150).

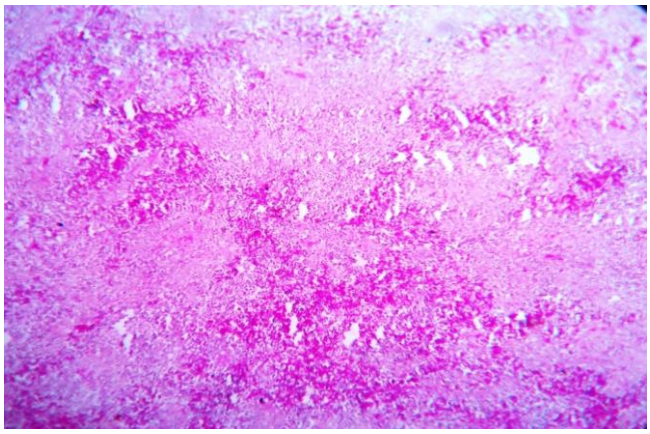

Fig 10: Section of spleen showing depletion of lymphocyte, necrosis and lymphocytolysis (H & E X 200).

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