

ISSN Print: 2664-9926 ISSN Online: 2664-9934 NAAS Rating (2025): 4.82 IJBS 2025; 7(9): 23-32 www.biologyjournal.net Received: 13-06-2025 Accepted: 15-07-2025

All the Author Details Below The references

Exploring the pathobiology of colibacillosis in broiler chickens reared in Ganderbal district of North Kashmir

Majid Shafi, Shabia Shabir, Omer Khalil Baba, Masood Saleem Mir, Shayaib Ahmad Kamil, Andleeb Rafiq, Zahoor Ahmad Wani, Yasir Afzal Beigh, Mudasir Ali Rather, Basharat Maqbool Wani and Showkat Ahmad Shah

DOI: https://www.doi.org/10.33545/26649926.2025.v7.i9a.473

Abstract

The aim of the study was to examine the Pathomorphological changes in the naturally occurring Colibacillosis in broiler chickens reared in commercial broiler farms at Ganderbal district of Kashmir. During this study, a total of 200 cases of Colibacillosis were identified at 10 different broiler farms and the dead birds were sent to the Division of Veterinary Pathology for a thorough postmortem investigation. Colibacillosis is a complex disease mainly occurring in the age group of 3-4 week age of age and is characterized by air sacculitis, pericarditis, peritonitis, salpingitis, synovitis, osteomyelitis or yolk sac infection. The pathological changes occurring in *Escherichia coli* infection were studied in natural cases of avian Colibacillosis. The samples were collected from different poultry farms of Ganderbal district of Kashmir. During postmortem examination, fibrin deposition on the liver and heart, as well as congestion in different organs were observed in the affected birds. The histopathological examination revealednecrosis and the depletion of lymphocytes in the spleen, interstitial pneumonia, fibrinous pericarditis, myocarditis, fibrinous perihepatitis, hepatitis and fatty degeneration in the hepatocytes. It is concluded that the pathogenic *E. coli* poultry led to systemic lesions and immunosuppression in birds.

Keywords: Escherichia coli, chicken, pathological lesions, colibacillosis

Introduction

Escherichia coli are thought to be the primary or secondary pathogen responsible for avian Colibacillosis, which is one of the main causes of morbidity and mortality in poultry (Lutful Kabir, 2010) [17]. The gas exchange regions of the lung and the air sacs are thought to be sites of entrance Escherichia coli into the circulation because they lack resident macrophages, making them relatively susceptible to bacterial invasion and colonization (Mellata et al., 2003) [19]. All age groups of poultry are susceptible to these diseases, but broiler chicks within the first four to six weeks of life are particularly vulnerable and seriously affected, with a high death rate (Leitner and Heller, 1992) [18]. Colibacillosis is a complex syndrome characterized by multiple organ lesions like air sacculitis, pericarditis, peritonitis, salpingitis, synovitis, osteomyelitis and yolk sac infection. The concomitant infection of E. coli and the turkey rhinotracheitis virus frequently causes swollen head syndrome in hens (Stehling et al., 2003) ^[25]. Colibacillosis is considered one of the main causes of morbidity and mortality in poultry worldwide. There are significant financial costs associated with respiratory tract infections, particularly the colisepticemia which is often followed by septicemia (Barnes and Gross, 1997 and Ewers et al., 2003) [4, 9]. Colisepticemia usually results in death, but certain birds can recover fully or only partially, resulting in meningitis, panopthalmitis, osteoarthritis, synovitis and coligranuloma characterized by multiple granulomas in the liver, caecum, duodenum, and mesentery (Barnes et al., 2003) [2]. Keeping in view the paucity of information regarding Colibacilosis in broiler chickens in Kashmir, this study was undertaken to evaluate the pathological alterations on the structure and functionality of different organs of infected broiler

Corresponding Author: Showkat Ahmad Shah Division of Veterinary Pathology, Faculty of Veterinary Sciences and Animal Husbandry, Sher-e-Kashmir University of Agricultural Sciences and Technology of Kashmir Jammu & Kashmir, India

Materials and Methods

• Study area and study period

The study was conducted at Division of Veterinary Pathology, F.VSc & AH, SKUAST-Kashmir.

• Selection of the cases

Both organized and unorganized poultry farms were visited regularly during the study period in order to record the mortality. During this study period, 200 mortality in broiler chicken were recorded due to Colibacillosis at 10 poultry farms that were the focus of our investigation. The outbreaks suspected for *Escherichia coli* in broiler chicken were identified based on the history, clinical signs and lesions, after following a thorough post mortem examination of birds.

• Relative organ weight ratio

The lymphoid organs like spleen, thymus, caecal tonsils and Bursa of Fabricius were removed from dead birds that had contracted Colibacillosis naturally. The relative weights of each of these organs were calculated using the method outlined by Garg (2004) ^[11].

• **Statistical analysis:** All the data generated in the study was analyzed using SPSS software as per Snedecor & Cochran, (1994) [26].

• Pathoanatomical studies

The carcasses were subjected to a thorough and systematic necropsy for examining and recording of the lesions true to colibacillosis which included perihepatitis, pericarditis, omphalitis, tenosynovitis, airsacculitis and cellulitis. The representative samples of liver, heart, lung, air sacs, kidney, intestines and spleen were collected from colibacillosis, subsequently preserved and fixed in 10% buffered formalin for histopathological examination and processed by routine paraffin embedding technique as described by Luna, 1968 [16].

Results

In this investigation, broiler chickens affected with naturally occurring colibacillosis in North Kashmir exhibited characteristic clinical signs and pathological changes. The observations have been presented under the following subheadings.

Clinical Manifestations: Birds infected with E.coli appeared weak, depressed and exhibit less activity as compared to normal birds. The E.coli affected birds shows reduced feeding ability and may have mucoid diarrhea leading to the dehydration with sunken eyes, dry skin and increased thirst. The respiratory symptoms such as coughing, sneezing, nasal discharge, labored breathing and dyspnea can be seen in severe *E. coli* cases. The *E.coli* infection in case of in laying hens can lead to a decrease in egg production and the production of misshapen eggs. Some cases of E. coli infection in birds can cause swollen joints leading to the lameness and difficulty in walking. The E. coli infection in birds can have severe consequences-like high mortality and morbidity rates, particularly in young birds resulting to the huge economic loses to the poor farmers. The birds with E. coli infections may also have reduced body weight, increased thirst, ruffled plumage and disheveled feathers. The infected breeding flocks may experience reduced fertility and may have substantial mortality

rates, especially in young chicks as compared to adult birds. Birds infected with E. coli may also exhibit neurological symptoms as paralysis, convulsions, and tremors. Some poultry flocks showed symptoms of disoriented movements, like stumbling and having trouble walking. The infected birds also tend to sit with drooping wings and have a hunched posture. The affected poultry birds may huddle up to stay warm. The infected birds at the end of study period may reveal pale mucus membranes with other different clinical manifestations like Tibial dyschondroplasia, aberrant bending of the tibial bones, enlarged hock joints, sternal recumbency, convulsion, eye lacrimation, excessive salivation, restlessness and ataxia. The keel bone appeared sharp and pointed due to emaciation. The infected birds may show significant increase in temperature beginning from the 1st day till end of the study period. The diseased birds also reveal stunted growth, watery droppings, weak muscles and a loss of reaction to stimuli before they died. The affected birds may reveal puffed feathers, closed eyes, stood motionless for long period of time. The bird's mouth appeared to be clogged with mucus and may also show repeated sneezing. Death usually resulted from respiratory failure. Before death, the birds fell suddenly revealing uneasiness, paddling of legs unconsciousness.

Pathomorphology

a) Heart

- **Gross Pathology:** Grossly, the heart showed signs of congestion and was covered with thick fibrinous layer in severe *E.coli* cases. The congestion in the heart results into fluid accumulation leading to edema. The heart of the affected birds appeared swollen and has a pale appearance.
- Histopathology: The pericardium was generally thickened and engorged with blood. In mild cases, heterophilic infiltration and fibrinous exudate results in the thickening of the pericardium. In addition to this, the histopathological analysis revealed also fibrinous pericarditis with congested blood vessel (Fig-1). In severe cases, there was an excessive thickening of the pericardium with necrotic regions and infiltration of heterophils. The microscopic examination further revealed severe myopathy, disruption of the heart muscle (Fig-2) and extensive leucocytic infiltration, primarily of heterophils visible in the spaces between the muscle fibers.

b) Liver

- Gross Pathology: The liver of the affected birds in moderate cases revealed deposition of fibrin on their surface. In severe cases, a thick layer of fibrin was covered the whole liver, giving it a "bread and butter" appearance. Some of these livers had a dark discoloration, appeared friable and had localized regions of necrosis visible on the surface.
- **Histopathology:** The microscopic examination of the liver of infected birds revealed degeneration in the form of cellular swelling, individualization of hepatocytes, deformed hepatic cords, congested blood vessel and Fibrosis (Fig-3). In some severe cases, kupfer cell hyperplasia and thickened liver capsule characterized by

the presence of a significant amount of fibrin, Sinusoidal congestion and infiltration of several heterophils in the affected tissue section (Fig-4).

c) Kidney

- **Gross Pathology:** In most Colibacillosis cases, the kidneys were enlarged, swollen and edematous in nature. On the surface of the kidneys, necrotic foci and pin-point hemorrhages were also found in severe *E.colicases*. At the end of the study period, the kidneys of affected birds were mottled with pale discoloration.
- **Histopathology:** The microscopic examination of the affected kidneys revealed congestion and hemorrhage in interstitial tissue accompanied with degeneration of renal tubules (Fig-5). In severe cases, the kidneys of infected birds revealed atrophy of the glomerulus and denudation of renal tubular epithelium along with focal infiltration of leucocytes consisting of both mononuclear cells and heterophils (Fig-6).

d) Lung

- **Gross Pathology:** The Pulmonary lesions ranged in severity from congestion, edema to lung consolidation. Some of the severe *E.coli* cases revealed patchy to diffuse areas of lung consolidation. In most instances, the consolidation was unilateral and in more advanced cases it was bilateral.
- **Histopathology:** Generally, the lung revealed parabronchial hemorrhages and interlobular septal congestion. The inflammatory cells like heterophils and mononuclear cells were found infiltrating the primary and the tertiary bronchioles lumen. There have been instances of acute bronchopneumonic changes, which were characterized by the infiltration of leucocytes and the presence of exudates in the bronchioles and parabronchi (Fig-7). The microscopic analysis of the lung further revealed thickened wall of the congested blood vessel as a result of inflammatory cells (Fig-8).

e) Air sacs

- Gross Pathology: The air sacs had a thin film of fibrin covering them in mild cases and a thick layer in advanced stages characterized by caseous exudates on the surface. The lesions were more pronounced on the thoracic air sacs as compared to the abdominal air sacs.
- **Histopathology:** The microscopic examination of the air sacs membrane revealed increased thickness in each case due to a leucocytic infiltration consisting predominantly of heterophils. The histopathological examination of the air sacs further revealed that the large amounts of fibrinous exudate and oedema were commonly pathological changes observed in the affected air sacs.

(f) Bursa

- Gross Pathology: In most cases of *E. coli* infection, the affected bursa showed lesions of edema, enlargement and swelling. The bursa of the affected poultry flocks were hemorrhagic with blood and cheesy exudate mixed together in the lumen. In addition to this, bursa of the affected birds was friable in nature with excessive amount of exudate and fibrin deposits in the bursal cavity.
- Histopathology: The histopathological examination of the bursa of Fabricius revealed lymphoid depletion,

which is characterized by a significant reduction in the quantity of lymphocytes within bursal follicles and the consequent loss of the bursal architecture (Fig-9). The further microscopic analysis revealed interstitial oedema, congestion, degeneration, atrophy and necropsy of the bursal follicles (Fig-10). As the disease progresses, the bursa undergoes further degeneration and the remaining lymphoid tissue may be replaced by connective tissue.

(g) Thymus

- **Gross Pathology:** Grossly, the thymus appeared smaller as compared to normal one, which could be the result of the *E. coli* infection-induced thymic atrophy and lymphoid depletion. The majority of the severe cases of Colibacillosis were found to have swelling, congestion and edema upon gross examination. In few *E.coli* cases, the thymus may become soft and fragile in nature.
- Histopathology: The histopathological analysis of the thymus showed depletion of lymphoid tissue resulting in decreased quantity of lymphocytes thymicatrophy (Fig-11). The lymphocyte depletion impairs the normal bird's immune system capacity to build a successful defense against *E.coli* infections. The microscopic examination of the thymus also showed infiltration of inflammatory cell, such as heterophils, macrophages and lymphocytes in response to the E.coli infection. In severe cases, microscopic examination further revealed degeneration and necrosis inside the thymus (Fig-12). These necrotic areas show that the bacterial cytopathic effects have resulted in localized cell death.

h) Spleen

- **Gross Pathology:** The spleen was enlarged and congested in the most of the *E.coli* cases. The scattered necrotic foci were also found on the spleen's surface in a few *E.coli* affected cases.
- **Histopathology:** The microscopic examination revealed severe splenic congestion, hemorrhages, lymphoid depletion (Fig-13) and many necrotic foci were also evident in the affected tissue section (Fig-14). In most of the severe cases, secondary germinal centers were formed as a result of extensive necrosis in the spleen's primary follicles. The histopathological examination of the spleen further revealed perivascular infiltration with thickening of the wall of the blood vessel.

I) Intestine

• Gross Pathology: The affected intestine has more vascularity and appears redder than normal because of vascular congestion. The gross examination of the intestine also reveals large hemorrhagic patches and pinpoint necrotic spots within the intestinal wall as a result of the hemorrhages in the affected intestinal mucosa. The *E.coli* infection can induce inflammation that thickens the intestinal wall and causes edema, which gives the appearance of swollen intestines. The affected intestinal mucosa consists of fibrin and debris which may appear as yellowish plaques. Sometimes narrowing of the intestinal lumen caused by fibrosis may also be observed in the affected intestine which can be resulted from inflammation and ulceration.

Histopathology: The microscopic examination of the intestinal mucosa revealed degeneration, denudation and necrosis. The affected intestinal villi showed blunting of the villus due to the presence of inflammatory cells (Fig-15). The microscopic examination further revealed glandular atrophy and fragmentation of intestinal villi (Fig-16). The microscopic analysis of ileum tissue section revealing denudation of tunica serosa with villous blunting (Fig-17). The histopathological examination of caecum revealing thickening of tunica muscularis with atrophy of the intestinal glands (Fig-18). The inflammatory cells like heterophils and lymphocytes can penetrate the intestinal lamina propria, intestinal glands and intestinal epithelium. The normal architecture of the intestinal epithelium may be disturbed by these heterophils.

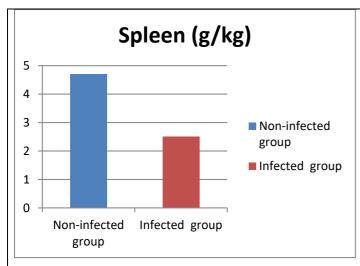
The mean weights of spleen, bursa, thymus and caecaltonsils from both infected and non-infected birds were comparable. On the other hand, when comparing the mean weights of various lymphoid organs in Colibacillosis-infected birds to those of normal birds, a significant ($P \le 0.05$) decrease was observed in the weight of lymphoid organs in *E.coli* infected bird. The decrease in the weight of the lymphoid organs must be due to the lymphoid depletion.

Table 1: Relative weight of lymphoid organs of broiler chickens infected with Colibacillosis

Groups	Spleen (g/kg)	Bursa (g/kg)
Non-infected group	$4.7^{a} \pm 0.13$	$3.30^{\circ} \pm 0.35$
Infected group	$2.50^{b} \pm 0.25$	$1.90^{d} \pm 0.13$

Means in different columns bearing common superscript does not differ significantly

• Relative organ weight ratio



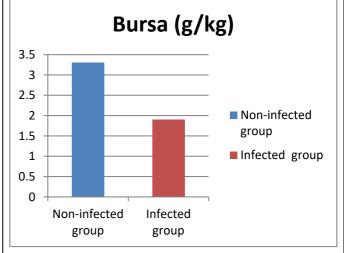


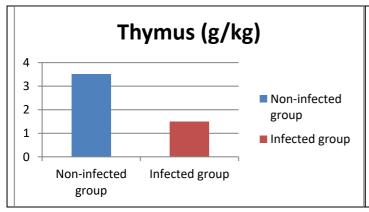
Fig 1A: Relative weight of Spleen and Bursa in Infected and Non-Infected Broiler Chickens

The graph represents alteration in relative weight of Spleen and Bursa in infected group and Non-infected group

Table 2: Relative weight of lymphoid organs namely Thymus and Caecal tonsils of broiler chickens infected with colibacillosis

Groups	Thymus (g/kg)	Ceacal Tonsils (g/kg)
Non-infected group	$3.5^{a} \pm 0.17$	$3.2^{c} \pm 0.27$
Infected group	1.5 ± 0.29	$1.6^{d} \pm 0.35$

Means in different columns bearing common superscript does not differ significantly



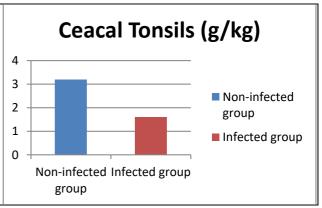


Fig 2A: Relative weight of Thymus and Caecal Tonsils in Infected and Non-Infected Broiler Chickens

The graph represents alteration in relative weight of thymus and caecal tonsils in infected group and Non-infected group

Discussion

The rearing of domesticated birds, such as chickens, ducks, turkeys, and geese, with the intention of generating meat, eggs, and other poultry products, is known as poultry farming (Singh *et al.*, 2018) ^[22]. It is an important sector of the agriculture and is essential to supplying animal protein to the world's population. Initially, poultry farming was primarily focused on egg production, but now it has been expanded to meat production as well. With a variety of production methods and management techniques, poultry farming is a highly specialized and productive industry. Poultry farming has several benefits, chief among them being its high productivity and short production cycle in comparison to other animals. Poultry farming has a significant economic impact, providing employment opportunities, income generation and food security.

The purpose of this experiment was to evaluate the pathomorpho logical and clinical alterations observed in the Colibacillosis infected broiler chickens. The clinical signs like depression, watery diarrhea, dehydration, ruffled feather, trembling, gasping and emaciation were recorded in *Ecoli* infection (Gangane *et al.*, 2006 and Shah *et al.*, 2019, Umar *et al.*, 2016 and Christie *et al.*, 1999) [10, 23, 27, 8].

The liver had extensive gross pathological lesions, including adhesions, rounding of the margins, congestion, and necrotic foci on the liver's surface. Microscopically, there might be the deposition of fibrin on the capsular wall heterophillic infiltration and kupfer cell hyperplasia (Kumar et al., 2013, Sharma et al., 2015 and Renu et al., 2012) [13, 24, 21]. An examination of the heart grossly revealed adhesions between the heart and the chest cavity, congestion, and varying fibrinous layer deposition on the pericardium. Cardiomegaly, involving both heart chambers, was observed during the gross examination of the heart. The microscopic examination revealed mononuclear cellular infiltration and fibrinous exudates in the affected heart tissue sections. These types of lesions have been previously described by few researchers in a natural outbreak of colibacillosis in broiler chickens (Nakamura et al., 1995, Jindal et al., 2003, Gangane et al., 2006 and Kumar et al., 2013) [20, 12, 10, 13]. Grossly the lungs revealed congestion and consolidation. The microscopic examination of the affected lungs revealed pneumonic changes as evident with infiltration of inflammatory cell in the lung tissue section with thickening of the wall of the blood vessel (Renu et al., 2012) [21]. In most cases, kidney lesions ranged from oedema and congestion to the development of tiny hemorrhages on the surface. The other pathological alterations like Congestion, hemorrhage, renal tubule degeneration, and localized infiltration of leucocytes of both mononuclear cells and heterophils were also observed in the damaged kidney tissue under microscopic analysis of the kidneys. These findings are in accordance with the results of other researchers (Baliar Singh et al., 1993, Christie et al., 1994 and Koynarski et al., 2010) [1, 8, 14] who also observed kidney edema and congestion in experimental colibacillosis (Gangane 2006 and Kumar *et al.*, 2004) [10, 15]. The fibrinous deposition on air-sacs could be attributed to severity of outbreak and an important pathological lesion in colibacillosis in broiler chickens (Renu et al., 2012) [21]. At the microscopic level, the primary and tertiary bronchioles' lumen revealed the presence of hemorrhages and congestion along with the infiltration of mononuclear cells and heterophils. Similar type of lesions has been observed in various Colibacillosis cases in broiler chickens which includes congestion, edema and pneumonic foci (Gangane et al., 2006, Christen et al., 1996, Tottori et al., 1997 and Kumar et al., 2013) [13]. The pathological lesions like organ atrophy and hemorrhagic lesions were visible on the surface of the bursa and other lymphoid organs. The microscopic examination of the lymphoid organs like spleen, bursa, caecal tonsils, and thymus, revealed lymphoid depletion leading to the immune suppression in the birds (Shah et al., 2019) [23]. In severe cases, a gross examination of the spleen revealed congestion and the presence of necrotic foci on the surface. The microscopic examination of the spleen showed lymphoid depletion, significant hemorrhages and congestion along with thickening of the blood vessel wall. The observations were in concurrence with the findings of various researchers published in reputed journals (Benjamin et al. 1998 and Kumar et al., 2013) [5, 13]. A significant reduction in relative weight of lymphoid organs were observed in the infected birds when compared with the weight index of lymphoid organs of normal birds (Berthault et al, 2018) [3]. The severe atrophy of the lymphoid organs and lymphocyte depletion in the lymphoid follicles of the bursa, spleen, thymus and caecal tonsils could be the cause of weight loss.

Gross lesions of Escherichia coli infected Broiler chickens





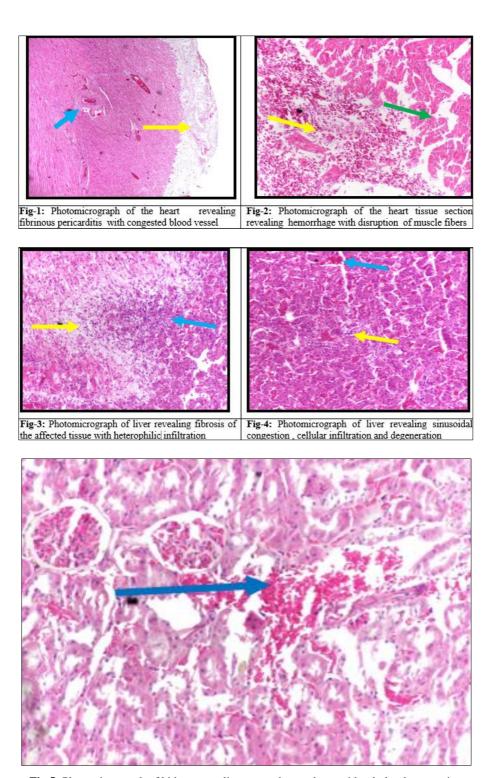


Fig A: Carcass of a broiler chicken showing Fibrinous perihepatitis and pericarditis



Fig B: Carcass of a broiler chicken showing Cloudy airsacs, Air saculitis and fibrinous peritonitis

Histopathology



 $\textbf{Fig 5:} \ \textbf{Photomicrograph of kidney revealing severe hemorrhage with tubular degeneration}$

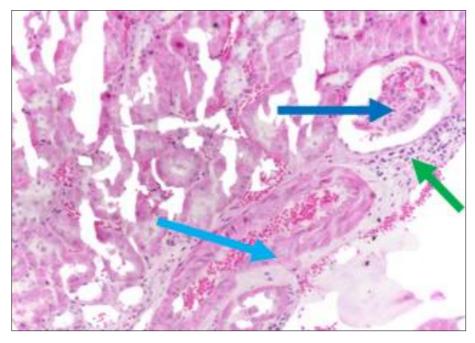
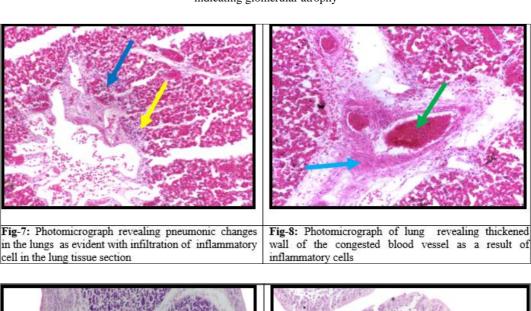


Fig 6: Photomicrograph of kidney revealing cellular infiltration, thick walled congested blood vessel with increased Bowman's space indicating glomerular atrophy



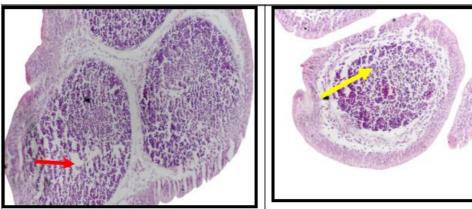


Fig-9:Photomicrograph of bursa revealing lymphoid depletion from bursa follicles

Fig-10:Photomicrograph of bursa revealing necrotic changes in the lymphoid follicles

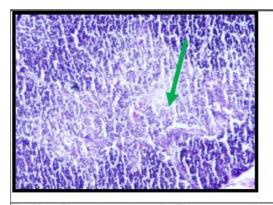


Fig-11: Photomicrograph of Thymus revealing lymphoid depletion from the lymphoid follicles

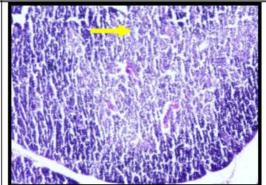


Fig-12: Photomicrograph of Thymus revealing necrosis within the thymic follicles

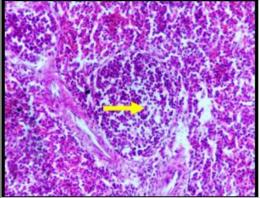


Fig-13: Photomicrograph of spleen revealing degeneration and destructed lymphoid tissue in the white pulp

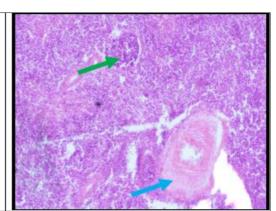


Fig-14: Photomicrograph revealing reveals splenic atrophy with thickening of the wall of blood vessel

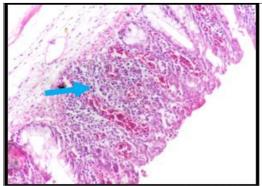


Fig-15:Photomicrograph of caecum revealing enteritis as evident with severe infiltration of heterophils in the affected tissue section

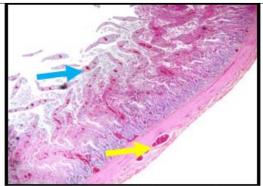


Fig-16: Photomicrograph of Jejunum revealing severe congestion with fragmentation of villi

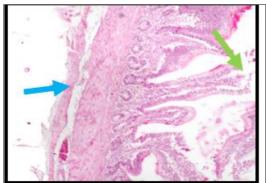


Fig-17:Photomicrograph of ileum revealing denudation of tunica serosa with villous blunting

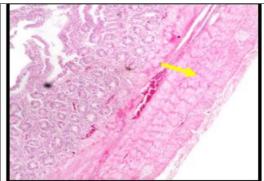


Fig-18:Photomicrograph of caecum revealing thickening of tunica muscular is with atrophy of the intestinal glands

Conclusion

The changes in the *E.coli* infected birds were seen in their gross and histopathological analyses. The pathomorphological alterations in the lymphoid organs including lymphoid depletion in the lymphatic follicles in spleen, bursa, thymus and caecal tonsils of infected broiler chickens which were suggestive of immunosuppressive and immunomodulatory effects of *E.coli* in broiler chickens.

References

- 1. Baliarsing K, Mishra R. Pathology of experimental colibacillosis in chicks. Indian Veterinary Journal. 1993;70:808-812.
- Barnes HJ, Vaillancourt JP, Gross WB. Newcastle disease in poultry. In: Saif YM, editor. Diseases of Poultry. 11th ed. Ames: Iowa State University Press; 2003. p. 631-656.
- 3. Berthault C, Larcher T, Denesvre C. Atrophy of lymphoid organs induced by *Escherichia coli* in broiler chickens. Veterinary Research. 2018;49(1):31-32.
- 4. Barnes HJ, Gross WB. Colibacillosis in poultry. In: Calnek BW, editor. Diseases of Poultry. 10th ed. Ames: Iowa State University Press; 1997. p. 131-141.
- 5. Benjamin M. Biochemical alterations of broiler chicken affected naturally with colibacillosis. Journal of Veterinary Clinical Pathology. 1998;12:43-47.
- 6. Christie G, Halliday W. Haematological and biochemical aspect of *Escherichia coli* septicemia in brown leghorn chicken. Avian Pathology. 1999;81:45-55.
- 7. Christen P, Metzler E. Changes in the transaminase enzymes in broiler chickens infected with *E. coli*. Avian Pathology. 1996;15:307-362.
- 8. Christie G, Halliday W. Hematological and biochemical aspect of *Escherichia coli* septicemia in brown leghorn chicken. Avian Pathology. 1994;81:45-55.
- 9. Ewers C, Janssen T, Wieler LH. Avian pathogenic *Escherichia coli* (APEC) in poultry birds. Wochenschr. 2003;116:381-395.
- 10. Gangane R, Kulkarni B, Yeotikar V. Studies on experimental colibacillosis in chicks. Indian Veterinary Journal. 2006;83:118-119.
- 11. Garg U, Asim K. Immuno-pathophysiological effects of colibacillosis in broiler chickens. Immunopharmacy. 2004;4:1709-1722.
- 12. Jindal N, Kumar A, Shukla L, Rottinghaus E. Effect of ochratoxin A on *Escherichia coli*-challenged broiler chicks. Avian Diseases. 2003;47:415-424.
- 13. Kumar A, Bhalerao D, Gupta P, Kumari M. Pathological studies on natural cases of colibacillosis in Haryana State. Haryana Veterinary Journal. 2013;52:118-120.
- 14. Koynarski V, Mircheva T, Karov S. Pathoanatomical and blood biochemical investigations in chicks challenged with *Escherichia coli* infection. Revue Veterinaire Clinique. 2010;161(3):133-144.
- 15. Kumar A, Jindal N, Ansari K, Rottinghaus G. Pathological changes in broiler chicken fed ochratoxins and inoculated with *Escherichia coli*. Avian Pathology. 2004;33(4):413-417.
- 16. Luna LG. Manual of Histologic Methods of Staining of Armed Forces Institute of Pathology. 3rd ed. New York: McGraw-Hill Book Company; 1968. p. 258.
- 17. Lutful Kabir SM. Avian colibacillosis and salmonellosis: A closer look at epidemiology, pathogenesis, diagnosis,

- control and public health concern. International Journal of Environmental Research and Public Health. 2010;7(1):89-114.
- 18. Leitner G, Heller ED. Colonization of *Escherichia coli* in young turkeys and chicken. Avian Diseases. 1992;36:211-220.
- 19. Mellata M, Dho-Moulin M, Dozois CM, Curtiss R, Lehoux B, Fairbrother JM. Role of avian pathogenic *Escherichia coli* virulence factors in bacterial interaction with chicken heterophils and macrophages. Infection and Immunity. 2003;71:494-503.
- Nakamura K, Yuasa N, Narita M. Effect of concurrent infection of *Escherichia coli* and infectious bursal disease in broiler chickens. Avian Pathology. 1995;19:713-721.
- 21. Renu S, Pruthi K, Londhe S, Anshu S. Etio-pathological studies on poultry mortality with reference to *Escherichia coli* infections. Indian Journal of Poultry Science. 2012;47:222-226.
- 22. Singh S, Taneja K. Competitiveness of Indian poultry industry in global context. Indian Journal of Animal Sciences. 2018;88:587-593.
- 23. Shah S, Mir M, Wani B, Kamil S, Goswami P. Pathological studies on avian pathogenic *Escherichia coli* infection in broilers. The Pharma Innovation Journal. 2019;8(7):68-73.
- 24. Sharma V, Jakhar K, Nehra V, Kumar S. Biochemical studies in experimentally *Escherichia coli*-infected broiler chicken supplemented with neem leaf extract. Veterinary World. 2015;8(11):1340-1345.
- 25. Stehling EG, Yano T, Brocchi M, da Silveira WD. Characterization of a plasmid-encoded adhesion of an avian pathogenic *Escherichia coli* (APEC) strain isolated from a case of swollen head syndrome. Veterinary Microbiology. 2003;95:111-120.
- 26. Snedecor GW, Cochran WG. Statistical Methods. 8th ed. Ames: Iowa State University Press; 1989.
- Umar A, Kamil S, Wani B, Mir M. Haematological and biochemical alterations of broiler chicken affected naturally with colibacillosis. International Journal of Current Microbiology and Applied Sciences. 2016;9(6):1906-1913.

Authors Details

Majid Shafi

Division of Veterinary Pathology, Faculty of Veterinary Sciences and Animal Husbandry, Sher-e-Kashmir University of Agricultural Sciences and Technology of Kashmir Jammu & Kashmir, India

Shabia Shabir

NIT-Kashmir

Omer Khalil Baba

Division of Veterinary Pathology, Faculty of Veterinary Sciences and Animal Husbandry, Sher-e-Kashmir University of Agricultural Sciences and Technology of Kashmir Jammu & Kashmir, India

Masood Saleem Mir

Division of Veterinary Pathology, Faculty of Veterinary Sciences and Animal Husbandry, Sher-e-Kashmir University

of Agricultural Sciences and Technology of Kashmir Jammu & Kashmir, India

Shayaib Ahmad Kamil

Division of Veterinary Pathology, Faculty of Veterinary Sciences and Animal Husbandry, Sher-e-Kashmir University of Agricultural Sciences and Technology of Kashmir Jammu & Kashmir, India

Andleeb Rafiq

Faculty of Veterinary Sciences and Animal Husbandry, Shere-Kashmir University of Agricultural Sciences and Technology of Kashmir Jammu & Kashmir, India

Zahoor Ahmad Wani

Faculty of Veterinary Sciences and Animal Husbandry, Shere-Kashmir University of Agricultural Sciences and Technology of Kashmir Jammu & Kashmir, India

Yasir Afzal Beigh

Faculty of Veterinary Sciences and Animal Husbandry, Shere-Kashmir University of Agricultural Sciences and Technology of Kashmir Jammu & Kashmir, India

Mudasir Ali Rather

Faculty of Veterinary Sciences and Animal Husbandry, Shere-Kashmir University of Agricultural Sciences and Technology of Kashmir Jammu & Kashmir, India

Basharat Maqbool Wani

Division of Veterinary Pathology, Faculty of Veterinary Sciences and Animal Husbandry, Sher-e-Kashmir University of Agricultural Sciences and Technology of Kashmir Jammu & Kashmir, India

Showkat Ahmad Shah

Division of Veterinary Pathology, Faculty of Veterinary Sciences and Animal Husbandry, Sher-e-Kashmir University of Agricultural Sciences and Technology of Kashmir Jammu & Kashmir, India