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Pathological findings in lungs and trachea of *Mycoplasma* infected broiler chickens in Haryana state

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ABSTRACT
Avian mycoplasmosis is one of the major problems faced by poultry industry all over the world. The present study was conducted on tissue samples of lungs and trachea collected from chickens suspected of mycoplasma infections during previous study of the department. The present communication reports the gross and histopathological findings in mycoplasma positive samples. For this, the pieces of lungs and trachea were fixed in 10% formalin and processed for histopathological examination as per the routine procedure. A total of 92 samples were subjected to present study. Gross pathological lesions observed at necropsy included severe congestion and consolidation of lungs with yellowish gelatinous exudates over the surface. Trachea revealed congestion and presence of fibrinous exudates in the lumen. Histopathological examination revealed marked congestion, haemorrhages, fibrinous exudate and infiltration of mononuclear cells, predominantly lymphocytes and heterophils in lungs and trachea. From this study it can be concluded, gross and microscopic alterations of the respiratory tract tissues alone are not sufficient to diagnose avian mycoplasmosis but these are suggestive of avian mycoplasmosis. However, histopathology is an adjunct diagnostic tool along with isolation, molecular techniques and immunohistochemistry.
Key words: Avian mycoplasmosis, trachea, lungs, histopathology

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INTRODUCTION

Mycoplasma gallisepticum (MG) and *Mycoplasma synoviae* (MS) are the two most pathogenic avian mycoplasmas that cause respiratory infection in poultry birds. *Mycoplasma synoviae* causes synovitis or mild upper respiratory disease whereas *M. gallisepticum* causes chronic respiratory disease (CRD) in chickens and have been reported to cause serious economic losses [1]. The clinical signs of avian mycoplasmosis are sneezing, coughing, snicks and respiratory rales; ocular and nasal discharge; decreased feed intake and egg production; increased mortality and poor hatchability. The increase in mortality in birds with MG infection caused by concurrent bacterial and/or viral infection has been widely reported [2, 3]. *Mycoplasma gallisepticum* infection has tropism primarily for mucosal membranes of the respiratory tract, conjunctiva and sinuses [4]. The organism usually enters the host via the respiratory tract except for the vertical transmission and upper airways and trachea are the preferred sites of infection for most of the strains of MG. Economic losses due to MG occurs due to increase in embryo mortality and chick mortality and due to a reduction in egg production, and a reduction in weight gain and feed conversion efficiency [5]. Although MG affects poultry industries worldwide, its pathogenesis of avian species is not well understood [6]. Previous research has indicated that attachment of MG to specific target cells via sialic acid residues along the respiratory epithelium is required prior to initiation of the disease processes and that a complex multifactorial process mediates cytoadherence [7]. The pathogenesis of MG is complicated by an organism's ability to alter its antigenic profile and thereby evade the host's immune system [8]. Also, mycoplasma ability to stimulate monocytes, macrophages, T-helper cells and NK cells, results in the production of substances, such as tumor necrosing factor (TNF-α), interleukin (IL-α, 2, 6) and interferon (α, β, γ) and these mechanisms may explain the transient suppression of humoral and cellular immune responses during mycoplasma infection in birds, the

immune tolerance and autoimmune diseases, as well as the massive lymphoid cell infiltration in the respiratory tract of infected fowls [9,10,11]. The pathogenicity of MG may be further complicated by the ability of MG to penetrate and survive within host cells resulting in dissemination throughout the host. Due to the above facts, the present study was conducted to observe the pathological changes of respiratory tract in poultry affecting with avian mycoplasmosis.

MATERIALS AND METHODS

Tissue Samples

A total of 92 tissue samples of lungs and trachea were collected from the chickens suspected for mycoplasmosis brought for necropsy examination to Disease Investigation Laboratory, Department of Veterinary Public Health and Epidemiology, Lala Lajpat Rai University of Veterinary and Animal Sciences, Hisar. A part of these samples was fixed in 10% neutral buffered formalin for histopathology examination.

Gross pathology

The detailed gross observations with specific lesions were recorded.

Histopathology

After proper fixation, the tissues were dehydrated in increasing grades of alcohol, cleared in xylene and embedded in paraffin blocks. Sections with 4-5µm thickness were subjected to haematoxylin and eosin for histopathological study as per conventional procedure [12].

RESULTS AND DISCUSSION

The present study reports the gross and histopathological lesions in 34 samples out of 92, of both lungs and trachea of chickens positive for mycoplasma infection which was confirmed by cultural isolation and PCR reaction in the other study conducted earlier [13]. Grossly, the lungs revealed severe congestion and consolidation with a yellowish accumulation of gelatinous exudates over the surface (Fig. 1). Trachea showed severely congested mucosa and presence of fibrinous exudates in the lumen (Fig. 2). Histopathological examination revealed marked congestion, haemorrhages, fibrinous exudate and infiltration of mononuclear cells, predominantly lymphocytes and heterophils in lungs (Fig. 3A and 3B). Sections of trachea showed marked deciliation, congestion, haemorrhages and infiltration of mononuclear cells predominantly lymphocytes and heterophils (Fig. 4A and 4B). The gross and microscopic findings in the present study were similar to that of earlier workers [14, 15]. Similar microscopic lesions were reported in chickens infected with avian mycoplasmosis which predominantly included congestion, thickening of tracheal epithelium with infiltration of lymphoid and plasma cells in the mucosa, massive infiltration of monocytes and heterophils in the lungs [14]. The lesions obtained in the present study could only be used as a suggestive factor of possible mycoplasma infection. These findings are supported by the results of isolation and PCR which detects and identify the causal organism. The present histopathological findings were in corroboration with another finding except for metaplasia of epithelial cells [15]. The more or less similar histopathological changes were observed by earlier workers in experimental studies [16, 17, 18]. From this study it can be concluded, gross and microscopic alterations of the respiratory tract tissues alone are not sufficient to diagnose avian mycoplasmosis but these are suggestive of avian mycoplasmosis. However, histopathology is an adjunct diagnostic tool along with isolation, molecular techniques and immunohistochemistry.

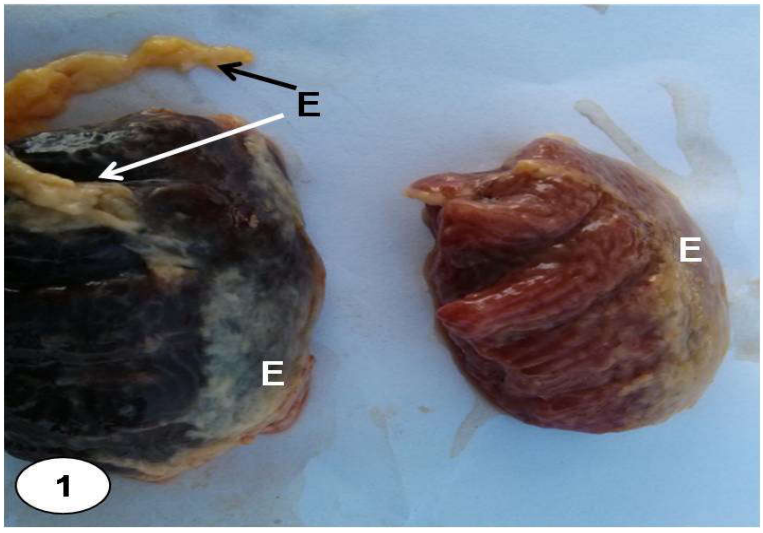

Fig. 1 Lungs showing severe congestion, consolidation and yellowish gelatinous exudate (E) over surface.

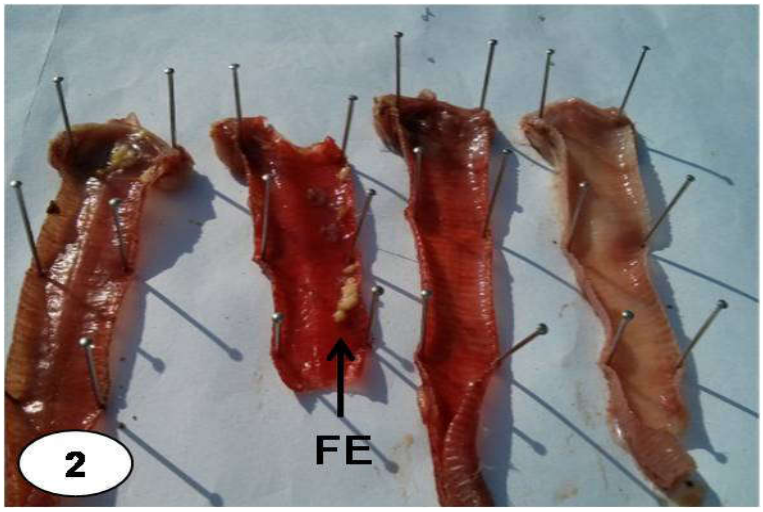

Fig. 2 Tracheal mucosa showing congestion and fibrinous exudate (FE) in lumen.

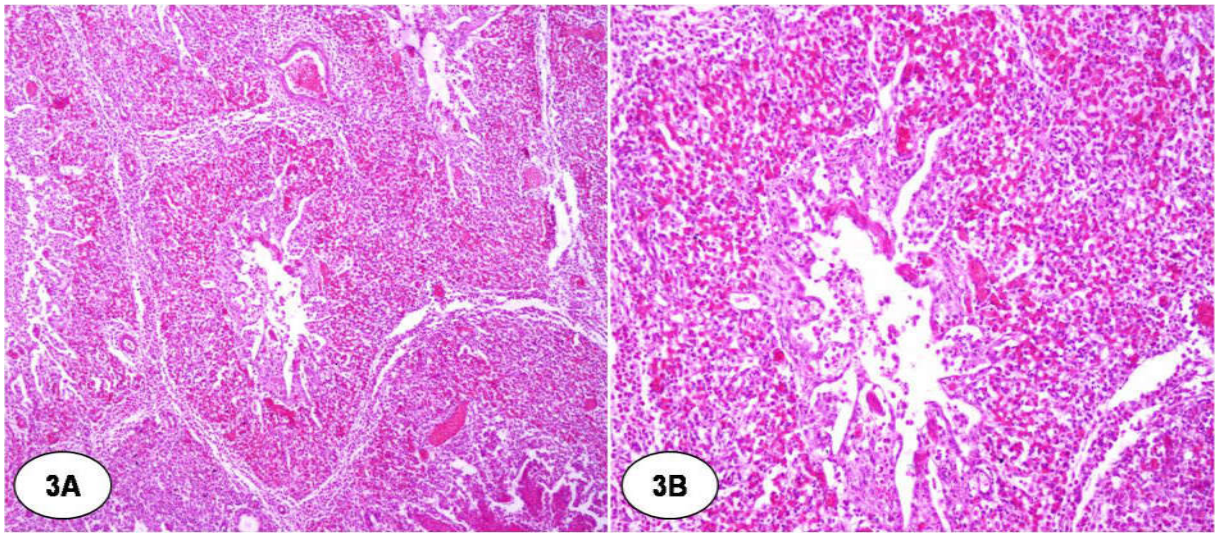

Fig. 3. 3A): Lungs showing marked congestion, haemorrhages and infiltration of mononuclear cells predominantly lymphocytes and heterophils. H&E×100 3B): Higher magnification of Fig. 3A. H&E×200

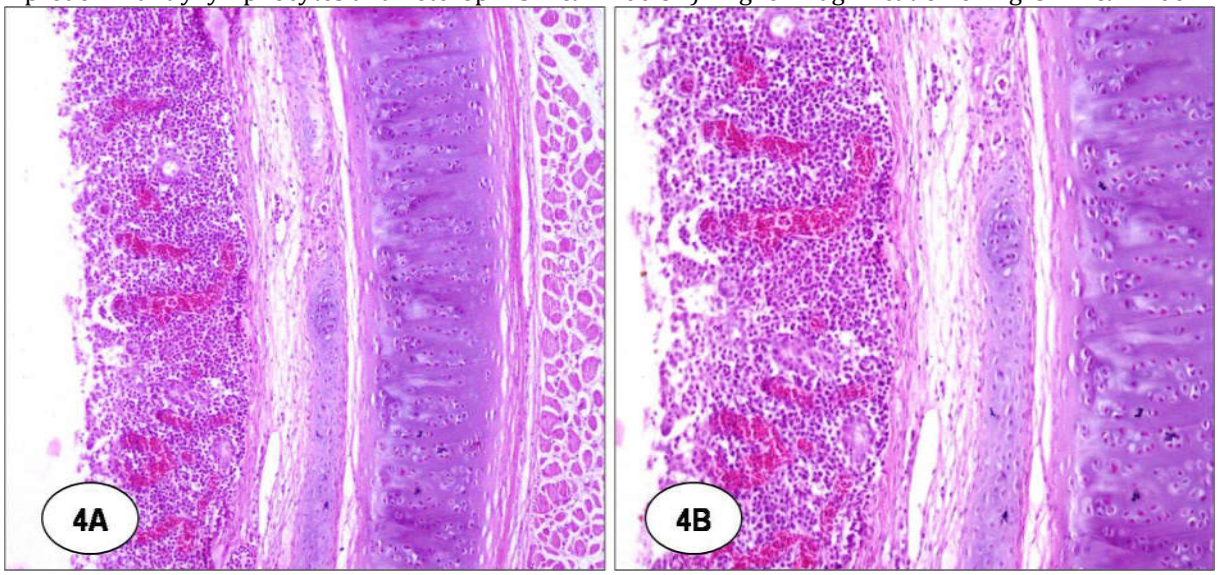

Fig. 4. 4A): Trachea showing marked deciliation, congestion, haemorrhages and infiltration of mononuclear cells predominantly lymphocytes and heterophils. H&E×100 4B): Higher magnification of Fig. 4A. H&E×200

REFERENCES

1. Osman, K.M., Aly, M.M., Amin, Z.M. & Hasan, B.S. (2009). *Mycoplasma gallisepticum*: an emerging challenge to the poultry industry in Egypt. Rev.Sci.Tech.O.I.E., 28(3): 1015-1023.
2. Bradbury, J.M. (2005). Poultry mycoplasmas: sophisticated pathogens in simple guise. Br Poult Sci., 46: 125-136.
3. Raviv, Z., Ferguson-Noel, N., Laibinis, V., Wooten, R. & Kleven, S.H. (2009). Role of Mycoplasma synoviae in commercial layer *Escherichia coli* peritonitis syndrome. Avian Dis., 51: 685-690.
4. Levisohn, S., & Kleven, S.H. (2000). Avian mycoplasmosis (*Mycoplasma gallisepticum*). Rev. sci. tech. Off. int. Epiz., 19: 425-442.
5. Kleven, S.H. (1990). Summary of discussions of avain mycoplasma team international research program on comparative Mycoplasmology, Hungary October 16–20, 1989. Avian Pathol., 19 (4): 795-800
6. Much, P., Winner, F., Stipkovits, L., Rosengarten, R. & Citti, C. (2002). *Mycoplasma gallisepticum* influence of cell invasiveness on the outcome of experimental infection in chickens. Immunol. Med. Microbiol., 34: 181–186.
7. Papazisi, L., Troy, K.E., Gorton, T.S., Liao, X. & S.J. Geary. (2000). Analysis of cytadherence-deficient, GapA-negative *Mycoplasma gallisepticum* strain R. Infect. Immun., 68: 6643–6649.
8. Levisohn, S., & Dykstra, M.J. (1987). A quantitative study of single and mixed infection of the chicken trachea by *Mycoplasma gallisepticum.* Avian Dis., 31: 1-12.
9. Razin, S. & Tully, J.G. (1995). Molecular and diagnostic procedures in mycoplasmology: molecular characterization. San Diego (CA): Academic Press; 1995. v.1.
10. Yamamoto, R. (1990). Mollicutes In: Biberstiein EL, Zee YC, editors. Review of veterinary microbiology. Chicago:Blackwell Scientific Publications,. p.213-27.
11. Razin, S., Yogev, D. & Naot, Y. (1998). Molecular biology and Pathogenicity of mycoplasmas. Microbiology and Molecular Biology Reviews., 62: 1094-156.
12. Luna, L.G. (1968). Manual of Histologic Staining Methods of the Armed Forces Institute of Pathology. (3rd edn.). McGraw Hill Book Company, New York.
13. Tomar, Piyush., Singh, Y., Mahajan, N.K., Jindal., Naresh & Singh, Mahavir. (2017). Molecular Detection of Avian Mycoplasmas in Poultry Affected with Respiratory Infections in Haryana (India). Int.J.Curr.Microbiol.App.Sci., 6(6): 2155-2162.
14. Barot, J. (2011). Cultural and molecular detection of avian mycoplasmosis. M.V.Sc theses submitted to IVRI, Izatnagar.
15. Stipkovits, L., Glavits, R. , Palfi, V. , Beres, A. , Egyed, L. , Denes, B., Somogyi, M. & Szathmary, S. (2012). Pathologic Lesions Caused by Coinfection of *Mycoplasma gallisepticum* and H3N8 Low Pathogenic Avian Influenza Virus in Chickens. Vet pathol., 49(2): 273-283.
16. Gaunson, J. E., Philip, C.J., Whithear, K.G. & Browning, G.F. (2000). Lymphocytic infiltration in the chicken trachea in response to *Mycoplasma gallisepticum* infection. Microbiol., 146: 1223–1229.
17. Bíró, Judit., Povazsán, J., Korösi, L., Glávits, R., Hufnagel, L. & Stipkovits, L. (2005). Safety and efficacy of *Mycoplasma gallisepticum* TS-11 vaccine for the protection of layer pullets against challenge with virulent *M. gallisepticum* R-strain, Avian Pathol., 34 (4): 341-347.
18. Gabriel, Senties-Cue., Shivaprasad, H.L. & Chin, R P. (2005). Systemic Mycoplasma synoviae infection in broiler chickens, Avian Pathol., 34(2): 137-142.

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