Iraqi Journal of Veterinary Sciences, Vol. 37, Supplement III, 2023 (65-72)
Proceedings of the 7th (2nd International) Scientific Conference, College of Veterinary Medicine, University of Al-Qadisiyah

Iraqi Journal of Veterinary Sciences

www.vetmedmosul.com

Monitoring of bronchiolar lesions and their relation with *Mycoplasma gallisepticum* and infectious bronchitis virus infections in broilers with severe respiratory distress

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Article information

Article history:
Received 01 December, 2022
Accepted 26 November, 2023
Available online 01 December, 2023

Keywords:
Bronchial lesion
Infectious bronchitis
Mycoplasma
Broilers

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Abstract

The goal of the study was to determine the relationship between incidences of chronic respiratory disease and infectious bronchitis disease in broiler in Nineveh province. One hundred ninety-eight broiler birds were isolated for this purpose after exhibiting confirmed severe respiratory disorders in 25 production fields from 1/11/2021 to 1/2/2022. The clinical observations were recorded, and blood was collected directly from the heart to separate serum for ELISA to confirm infection with *Mycoplasma gallisepticum* and infectious bronchitis virus antigens, and histopathological examination. The results demonstrate a significant variation in incidence rate of the infection between the chronic respiratory disease by *Mycoplasma gallisepticum* reaching 81.3%, were the incidence rate of infection with infectious bronchitis virus was 1.5 %, the incidence rate of the mixed infection was 1.5% as well. The gross examination also supported the clinical signs by revealing pathological changes with an inflammatory nature in all the examined birds, including pulmonary congestion, fibrin deposition, adhesions between pulmonary lobs, and air sacs. Caseous exudate deposition in bronchi and air sacs is also seen. The study revealed the presence of histological lesions in all three monitoring foci in the avian lung; the most frequent lesions were the caseous necrotic bronchitis and bronchiolar obstruction by caseous exudate at ratios 76.76 and 72.72%, respectively, at the pulmonary bronchi and peribronchiolar area. These lesions were variated in their severity between samples. The correlation test results showed a significant link between infection with *Mycoplasma gallisepticum* and bronchiolar epithelial desquamation at significance level. It has been concluded that the incidence of lesions was more synchronized and correlated to the infection of *Mycoplasma gallisepticum.*

DOI: 10.33899/ijvs.2023.137189.2649,

Introduction

Chronic respiratory disease in chickens is characterized by severe respiratory symptoms (1). Also, it causes significant economic losses to the poultry industry by reducing egg production, hatchability, and spoilage of carcasses (2). It causes a group of microorganisms called the collection of pleuropneumonia group, the most prominent of which are *Mycoplasma gallisepticum* and *Escherichia coli*. This disease does not often occur alone, as it may be associated with several diseases, including Newcastle disease, infectious bronchitis, chronic fowl cholera, and other bacterial diseases. Therefore, this disease is a mixture of different conditions and often causes high mortality rates in broiler chickens and economic losses. Significant due to low weight gain, isolation of infected birds, and their culling, especially when an outbreak of air sac infection, as well as in laying hens, leads to a sharp decrease in egg production

up to 30-40% (1). Infectious bronchitis disease is present in broiler chickens, and it is common and of commercial importance alike, as it causes death, growth retardation, and high rates of spoilage of broiler birds, as all these reasons lead to economic repercussions in the poultry industry. Moreover, there is a decrease in the production of Eggs, internal and external egg quality, and hatchability in laying hens and Breeder mothers. Secondary pathogens can make the treatment of this condition more difficult, which leads to higher rates of infection and death, as the infection reduces the weight gain of infected incubating chickens as well as the quality of eggs and infected laying hens' outputs, which leads to economic effects (3,4). Several common diseases can affect poultry's respiratory tract (airways, lungs, and alveoli). These include Newcastle disease, infectious bronchitis, avian influenza, laryngitis tracheotracheitis, Mycobacterium bolus, chronic respiratory disease, contagious sinusitis, and mycoplasma (5). The designed project detects respiratory diseases among chickens based on their abnormal sounds, such as coughing and grunting. The system was modeled using various audio sensors and hardware components, a microcontroller, a temperature sensor, and a liquid crystal display. The system works so that the sound sensor gets sound from the chicken's environment and sends it to the microcontroller, which interprets the input. The farmer will be notified if there is an abnormal sound, such as coughing or snoring. This systems architecture is based on the context of operations and processes in real-time scenarios (5). The researcher Al-Yadav *et al.* revealed that respiratory infections are a significant concern for the poultry industry. This study aimed to determine the extent of respiratory viral and bacterial co-infection with avian respiratory plasma in poultry flocks (6). Microscopic pathology in chickens and turkeys induced by *Mycoplasma gallisepticum* infection is characterized by substantial thickening of the mucous membranes of afflicted respiratory tract tissues as a result of infiltration with mononuclear cells, mainly lymphocytes and lymphoid follicle hyperplasia (7-9). Metaplasia of the respiratory epithelium has been documented, transitioning from pseudostratified ciliated columnar to non-ciliated low cuboidal or squamous (8). Tracheal mucosal thickness is widely used to assess the severity of MG illness (8,10).

The main target of the study was to determine the relationship between incidences of chronic respiratory disease and infectious bronchitis disease in broiler with severe respiratory disorders in broiler production fields at various locations in Nineveh province.

Materials and methods

Survey

Twenty-five broiler production fields were visited for selecting birds with severe respiratory distress; these fields were located in various regions of Nineveh province, including Rbiaa, Hamdanya, Bashiqa, Gogjali, Bartella, Telqaif, Quiara, Sherqate, Humaidate, and Telaafer, from 1/11/2021 to 1/2/2022.1Ninety-eight broilers were selected from the visited fields, showing severe clinical signs of respiratory distress; the clinical symptoms were also recorded, and the birds were transported to the laboratory for sampling.

The blood and tissue samples

The blood is collected directly from the heart to a clean plastic tube, left to solidify, and centrifuged at 2000 RPM for 5 minutes to separate serum, which is collected and kept in deep freeze. Birds were euthanized by cervical dislocation. The necropsy findings were recorded, the lower respiratory system was taken, and it was preserved in 10% neutral buffered formalin.

Serological confirmation for pathogens

Both *Mycoplasma gallisepticum* and Infectious bronchitis virus antigens were detected by estimating antibody levels in serum using the indirect ELISA technique. The ELISA kit from Biocheck was used; the conjugated antibody reacted to the antigen in the plate that wasn't trapped by serum antibodies, and the absorbance was assessed at 650 nm wavelength and calibrated to antibody titer (11). The vaccine index (VI) was calculated for each field (11). The titer of samples from the same area was compared to the site VI, and the values above were considered as infected; the infected - noninfected characteristics were turned to the digital values 1 and 0 for the two pathogens to be used for statistical analysis (12).

Histopathological examinations

Ninety-nine lower respiratory tract tissue samples were selected randomly and processed for microscopical examination through trimming, dehydration, clearing, embedding with paraffin, sectioning, and staining with hematoxylin and eosin (13,14). The microscopical test for each sample focused on the bronchi, bronchioles, and peribronchiolar areas. The monitored pathological changes were recorded, and their severity was expressed digitally to 4 grades as 0, 1, 2, and 3 to determine the lesion score for each sample (14,15).

Statistical analysis

The relation between each pathogen infection status and the occurrence of each noticed lesion with severity was estimated using Spearman's correlation test for ordinal data / SPSS /Version 19 (12).

Results

Clinical signs

The clinical signs included sternal or lateral recumbency, Nasal discharge, extended neck with gasping, lacrimation,

Moist rales sound with breathing, sneezing, and difficulty breathing.

Necropsy Findings

The lungs showed mild to severe congestion with regions of hepatization; most of the bronchi are inflamed, containing either frothy catarrhal exudate or caseated purulent exudate, which may obstruct the lumen, fibrinous exudate noticed in many samples covering the surface of the lungs, air sacs, and liver surface in the form of fibrinous pseudo membranes, air sacs contained caseation (Figures 1-4).

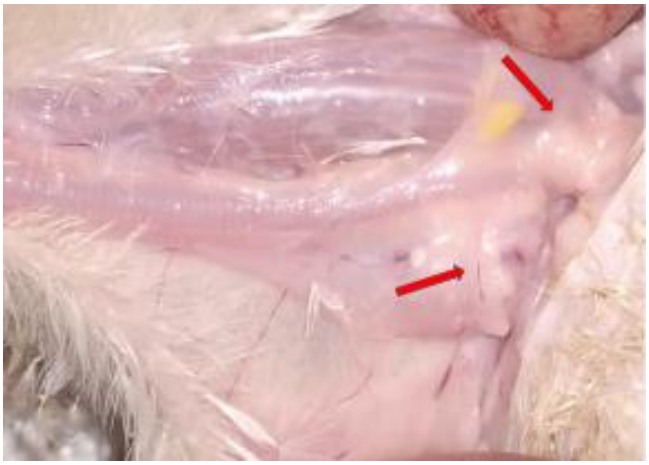

Figure 1: The tracheal bifurcation is distended with exudate (Red arrow).

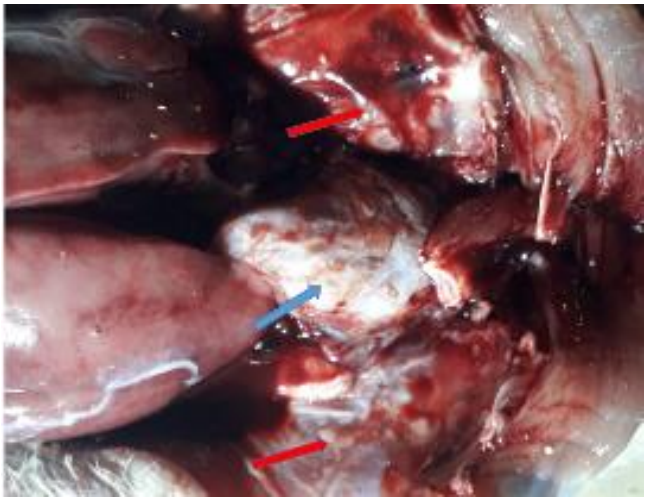

Figure 2: Pulmonary congestion and hemorrhage (Red arrow) with fibrinous exudate covering the pericardial surface (Blue arrow).

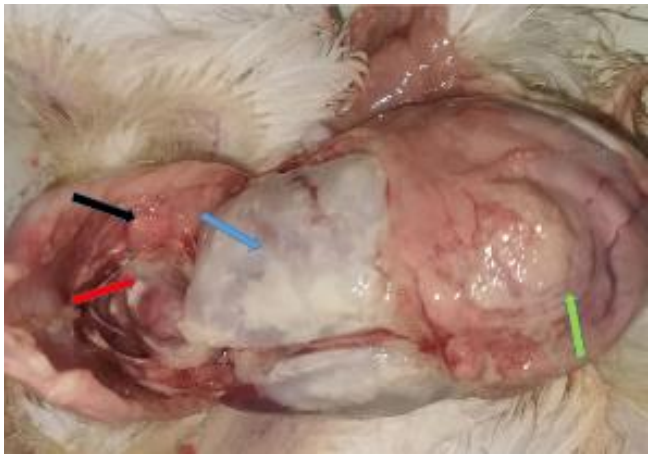

Figure 3: Fibrinopurulent exudate precipitated on the liver surface (Blue arrow), Pericardial sac (Red arrow), lung surface (Black arrow), and between abdominal viscera (Green arrow).

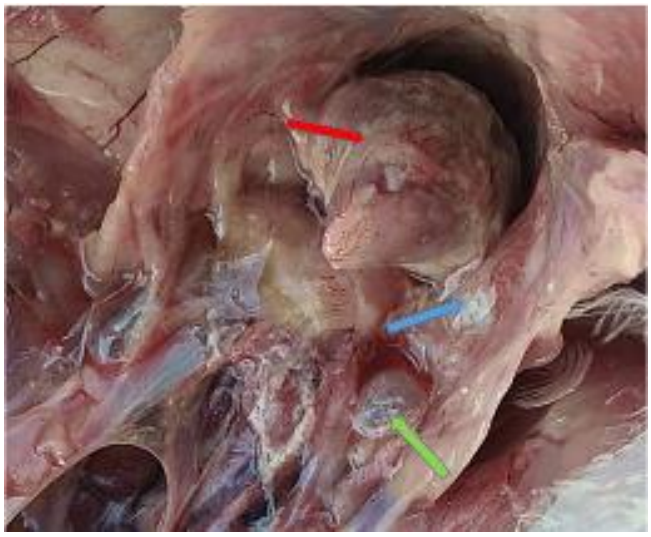

Figure 4: Sever pulmonary congestion (Blue arrow) with purulent exudate in pericardium (Red arrow) and air sacs (Green arrow).

Serological identification

ELISA test results for all studied samples Infection with the infectious bronchitis virus was cleared in only three birds, with a 1.5% incidence rate from the total samples. While infection with *Mycoplasma gallisepticum* was confirmed in 161 birds with an incidence rate of 81.3%, dual disorders appeared in 1.5% of actual examples (Table 1).

Table 1: Incidence rates of infectious bronchitis virus and *Mycoplasma gallisepticum* in broilers

Field No	No isolated birds	IBV positive%	MG positive%	Mixed infection%
1	4	0%	75%	0%
2	3	0%	35%	0%
3	5	20%	100%	20%
4	7	0%	100%	0%
5	5	0%	60%	0%
6	4	0%	100%	0%
7	8	0%	100%	0%
8	5	0%	100%	0%
9	26	0%	100%	0%
10	15	13.3%	100%	13.3%
11	17	0%	100%	0%
12	8	0%	62.5%	0%
13	2	0%	50%	0%
14	5	0%	100%	0%
15	10	0%	90%	0%
16	5	0%	100%	0%
17	4	0%	100%	0%
18	4	0%	100%	0%
19	5	0%	100%	0%
20	13	0%	30.7%	0%
21	10	0%	100%	0%
22	4	0%	75%	0%
23	10	0%	70%	0%
24	4	0%	75%	0%
25	3	0%	66.6%	0%
	Total number of samples	Total incidence of IBV	The total incidence of MG	Total Mixed infection
	198	1.5%	81.3%	1.5%

Histopathological examination

Pathological changes under the microscope were noticed at variable degrees of severity; those included acute catarrhal bronchiolitis at 17.17% of the examined tissues (Figure 5), Necrotic bronchiolitis with caseation at 76.76% of sections (Figure 6), with bronchi and bronchiolar obstruction with caseous exudate at 72.72% of samples (Figure 7). Submucosal hemorrhage appeared in 26.26% of samples (Figure 8), bronchial atelectasis was noticed in 12.12% of examined pulmonary tissues (Figure 9), and peribronchiolar fibrosis at 31.31% of the total samples (Figure 10, Tables 2 and 3).

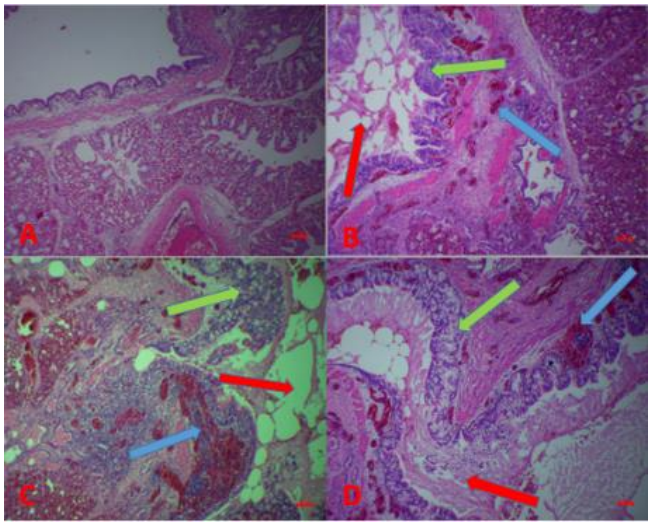

Figure 5: Normal avian bronchial histology (A), Acute catarrhal bronchitis including Catarrhal exudate (Red arrow), Bronchial vascular hyperemia (Blue arrow), and Mucinous degeneration of bronchial epithelium (Green arrow). The lesion in grades screened in images as (A=0), (B=1), (C=2), (D=3). Scale bar=50 µm. Staining H&E. Magnification=40X.

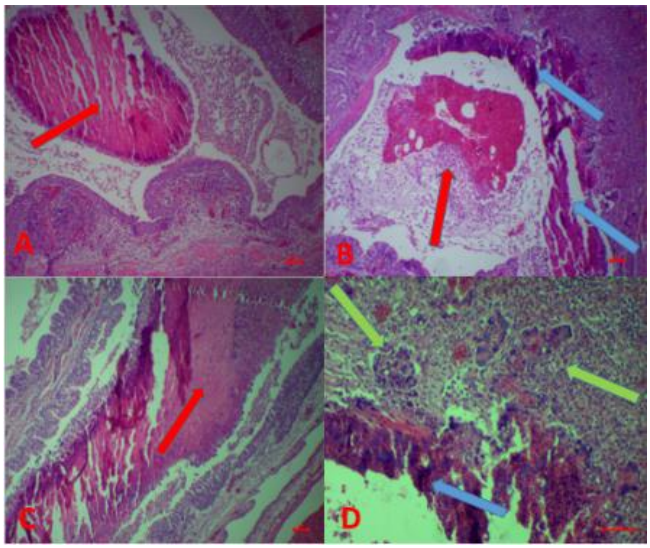

Figure 6: Caseous necrotic bronchitis including Caseous exudate in bronchial lumen (Red arrow), Necrotic bronchial epithelium (Blue arrow), Granulomatous inflammatory reaction in bronchial walls with presence of giant cells and macrophages (Green arrow). The lesion in grades screened in images as: (A=1), (B=2), (C=3) Magnification 40X, D=Magnified Image 100X. Scale bar=50 µm. Staining H&E.

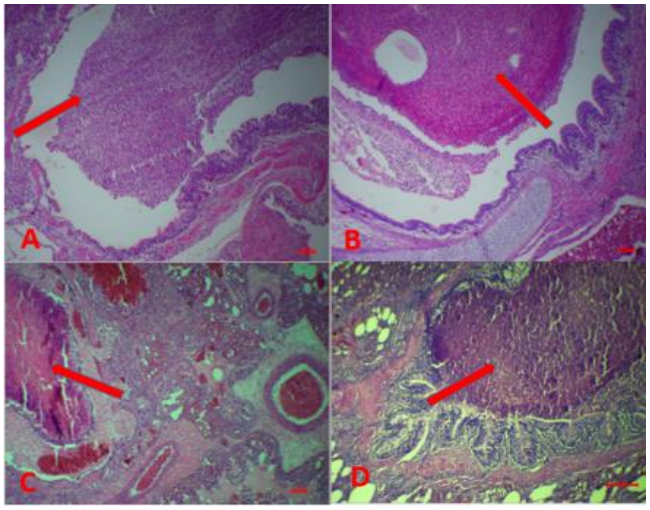

Figure 7: Bronchiolar lumen obstruction with caseated exudate (Red arrow). The lesion in grades screened in images as: (A=1), (B=2), (C=3) Magnification 40X, D=Magnified Image 100X. Scale bar=50 µm. Staining H&E.

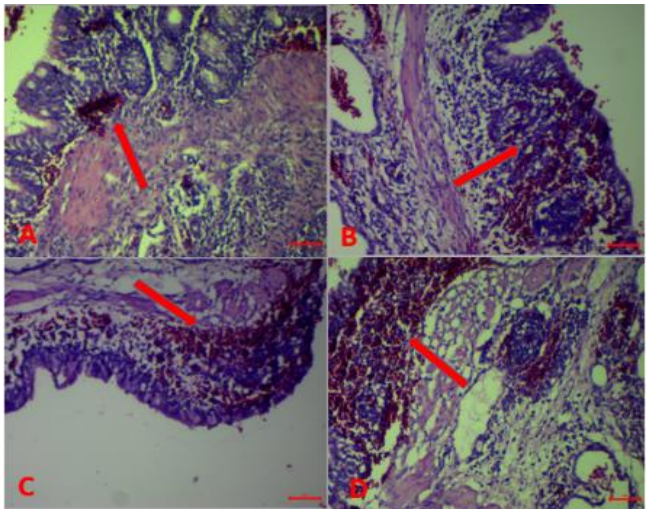

Figure 8: Bronchial submucosal hemorrhage (Red arrow). The lesion in grades screened in images as (A=1), (B=2), (C=2), (D=3). Magnification 100X. Scale bar=50 µm. Staining H&E.

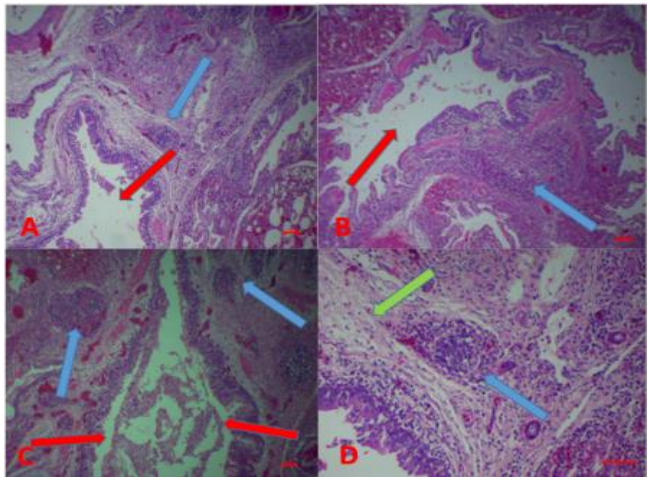

Figure 9: Chronic bronchitis with bronchial atelectasis, including irregular dilation of the bronchial lumen (Red arrow), Focal infiltration of mononuclear inflammatory cells (Blue arrow), and fibrosis in the bronchial wall (Green arrow). The lesion in grades screened in images as: (A=1), (B=2), (C=3) Magnification 40X, D=Magnified Image 100X. Scale bar=50 µm. Staining H&E.

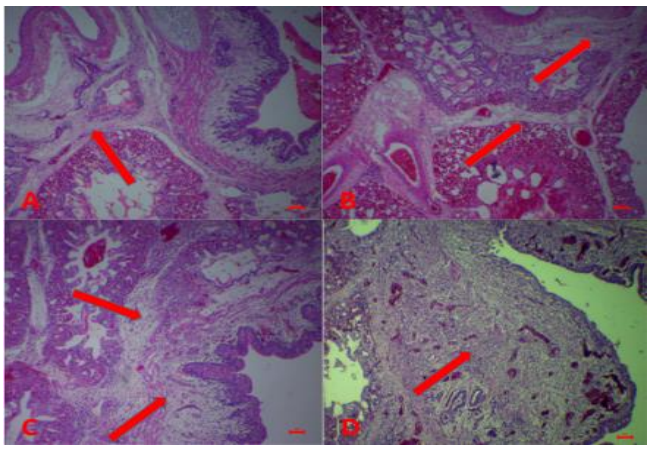

Figure 10: Peribronchial fibrosis or peribronchial fibroplasia (Red arrow). The lesion in grades screened in images as (A=1), (B=1), (C=2), (D=3). Magnification 40X. Scale bar=50 µm. Staining H&E.

Table 2: The recorded histological lesions with their Incidents at the bronchiolar and peribronchiolar area in broilers' lungs

No	Lesion	Number of samples	Incidence rate
1	Acute catarrhal bronchitis	17	17.17%
2	Caseous necrotic bronchitis	76	76.76%
3	Bronchiolar obstruction with caseous exudate	72	72.72%
4	Bronchiolar submucosal hemorrhage	26	26.26%
5	Bronchial atelectasis	12	12.12%
6	Peribronchiolar fibrosis	31	31.31%
A total number of the histologically examined samples=99 samples			

Table 3: Spearman's correlation coefficient values evaluating the link between *Mycoplasma gallisepticum* and IBV infections and the severity of histological lesions at bronchioles and peribronchiolar area of broilers

Lesions	MG		IBV	
	R	Sig.	R	Sig.
Acute catarrhal bronchitis	-0.037	0.716	0.091	0.373
Caseous necrotic bronchitis	0.043	0.676	0.197	0.051
Bronchiolar obstruction with caseous exudate	0.060	0.554	0.163	0.107
Bronchiolar submucosal hemorrhage	0.004	0.971	0.021	0.836
Bronchial atelectasis	-0.093	0.358	0.107	0.291
Peribronchiolar fibrosis	-0.038	0.710	0.173	0.087

R=Correlation coefficient, Sig=*(significant at P≤0.05) **(significant at P≤0.01)

Discussion

Histopathological examination of the bronchioles and their surroundings showed inflammatory pathological changes, as acute catarrhal bronchiolitis, necrotic bronchiolitis, and bronchiolitis with inflammatory caseous exudate were observed. These changes accompany many respiratory infections in poultry, such as the respiratory form of Newcastle disease, infectious bronchial disease, Infectious Laryngotracheitis, and Avian influenza, which all cause acute catarrhal bronchiolitis; this has been proven by some sources (16). According to clinical observations, it has been mentioned that influenza usually affects only the trachea and the main bronchi. The mucus is abundant, sometimes yellow, in the lower parts of the trachea and the central airways. This secretion may derive from glands connected to the mucous membrane, particularly numerous in the trachea and the main bronchi (13). The epithelium itself may produce mucus by a drastic change in which almost every columnar cell is converted into a goblet cell (13). There are no other changes in uncomplicated cases; There is no penetration or destruction of the wall elements, and the rule is to return things to normal. Bronchial cold sometimes occurs in the middle and small airways (13). These tubes are poorly supplied with glands attached to the mucosa, so converting columnar cells into goblet cells is the primary source of mucus. This process is better observed in recent cases of asthma, where the catarrh spreads to the small and bronchial airways (13). in 1981the researchers, Charlier *et al.* (13) added that the most critical lessons in the acute level of the disease are catarrhal inflammation in the respiratory pathways such as the nose, trachea, bronchi, and air follicles, where their walls become thick. The secretions are yellow-white, and the most essential characteristic is sinusitis and thickening in the walls of the air sacs. It may contain purulent materials or catarrhal and straightforward pneumonia. In severe cases, the fibrositis in the air sacs is acute with inflammation of the liver and its capsule, and a fibrinous layer is formed around the liver and the heart with severe inflammation of the pericardium (13).

Bronchial atelectasis and fibrosis around the trachea are inflammatory changes that are subacute or chronic and often accompany mycoplasma bacteria. Some sources have mentioned (17). The above said that in some cases, the accumulation of exudate can be severe enough to obstruct the bronchioles of the corresponding lobes. Supportive bronchopneumonia may be associated with mild fibrinous pleurisy. In addition, lung failure and collapse are reported to occur due to edema and pulmonary fibrosis, and peripheral acute pulmonary congestion is often seen in animals euthanized by barbiturates (medications that cause you to relax or feel drowsy). In addition, the researchers emphasized the Idiopathic pulmonary fibrosis, also known as idiopathic fibrotic alveolitis, is one of a family of idiopathic pneumonia that shares the clinical features of dyspnea, diffuse pulmonary infiltrates evident on X-rays, and varying degrees of inflammation, fibrosis, or both on biopsy (18). Previous studies included several forms of idiopathic interstitial pneumonia under idiopathic pulmonary fibrosis. Still, today, the clinical designation idiopathic pulmonary fibrosis should be reserved for patients with a specific form of fibrotic interstitial pneumonia referred to as Usual interstitial pneumonia. These infections are accompanied by bleeding and desquamation of the bronchial epithelium. This has been mentioned by Ansari *et al.* during their studies; He noticed a pronounced disruption and desquamation of ciliated epithelial cells and a significant decrease in goblet cells in the tracheal epithelium stimulated by intraperitoneal LPS (19). According to age and season, the current study looked at flock mortality owing to LPAI and concurrent infections with IB, MG, MS, and *E. coli*. When compared to previous findings, flock-wide mortality owing to LPAI (20,21).

The tracheal lesions identified during the current outbreaks, such as mucosal congestion with caseous exudate forming plugs at the tracheal bifurcation and spreading into the lower bronchi (22-24), and indicative of particular LPAI lesions. Histopathological abnormalities detected in the trachea and bronchi, such as denudation of epithelial linings and fibrin necrotic mass, were consistent and had previously been documented by other researchers (20,23,25). The lung lesions, such as vascular engorgement, hemorrhages, and fibrinous exudate in the parabronchi and heterophil and lymphocyte infiltration, were identical to those described

previously (25,26). Complications of these infections often occur with the invasion of pyogenic bacteria, which causes suppuration of the bronchial epithelium, caseous necrosis, and leads to blockage of the bronchioles with caseous inflammatory exudate (27). In the current study, histological examinations of morbid tissues from positive cases confirmed by PCR, such as the trachea, lungs, heart, and liver, were performed to record alterations at the cellular level. The trachea and lungs showed the most consistent histological changes. The epithelium of the trachea was necrosed and enlarged. Some places have a thick coating of mucus covering epithelial cells and cilia. Leukocytes infiltrated the epithelial and submucosal layers. The thickness of the epithelial mucous glands increased due to cellular infiltration, and edema mucosal sloughing and hemorrhages of varying severity were also seen, correlating with the findings of Gaunson *et al.* (28).

Lungs exhibited congestion, hemorrhages, localized necrosis, and leukocytic infiltration (lymphocytes and polymorphs). Emphysema and exudates in the alveoli were also seen. Grey hepatization was also seen in slides of specific lungs. Saif *et al.* also detected giant cells and military granuloma in the lungs (29). In severe instances involving *E. coli*, the liver, and the heart, they revealed congestion and leukocytic infiltration (Lymphocytic and polymorphonuclear). Hepatocyte hemorrhages, degeneration, and necrosis were all observed in certain sections. There was also heart muscle degeneration and pericarditis (30).

Conclusion

It has been concluded that the incidence of lesions was more synchronized and correlated to the infection of *Mycoplasma gallisepticum. At the same time,* minimal pathological relevance was linked with the infectious bronchitis virus, which may be due to the success of the applied vaccination programs to control it in fields.

Acknowledgment

Thanks to the College of Veterinary Medicine/University of Mosul and the pathology and poultry disease department for their support.

Conflict of interest

We declare no conflict of interest related to the article.

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رصد أفات الشعب الهوائية وعلاقتها بالإصابة بالمايكوبلازما الدجاجية الأنتانية وفايروس التهاب الشعب الهوائية المعدي في الفروج المظهر للكرب التنفسي الشديد

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الخلاصة

هدفت الدراسة الى معرفة الارتباط بين حدوثية كل من المرض التنفسي المزمن ومرض التهاب الشعب الهوائية في طيور فروج اللحم. لهذا الغرض تم عزل ١٩٨ فروج لحم شخص سريريا بظهور العلامات التنفسية الشديدة من ٢٥ حقلا للفترة من ٢٠٢١/١١/١ الى ٢٠٢٢/٢/١. سجلت العلامات السريرية وتم سحب الدم منها وعزل مصل الدم لإجراء اختبار الأنزيم المناعي الممتز لكل من مستضد المايكوبلازما الإنتانية الدجاجية ومستضد فايروس التهاب الشعب الهوائية المعدي. أظهرت النتائج وجود تباين كبير في نسبة الاصابة، فقد وصلت نسبة الاصابة بالمرض التنفسي المزمن الذي تسببه المايكوبلازما الدجاجية الإنتانية الى ٨١,٣% في حين لم تتجاوز نسبة الاصابة بمرض التهاب الشعب الهوائية ١,٥%.و كانت نسبة الاصابة المشتركة بالمرضين المدروسين ١,٥% فقط في فروج اللحم المظهر لعلامات القصور التنفسي الشديد و ثبتت نتائج التشريح المرضي العياني وجود الآفات المرضية ذات الطبيعة الالتهابية في جميع طيور فروج اللحم المظهر للعلامات السريرية تمثلت ابرزها بالاحتقان الرئوي و ترسب طبقات من الليفين والالتصاقات بين الفصوص الرئوية لقد بينت الدراسة وجود الآفات المرضية النسيجية المتنوعة كان ابرزها التهاب القصيبات التنخري وانسدادها بالنضحة الالتهابية بالنسب ٧٦,٧٦ و٧٢,٧٢% على التوالي في منطقة القصيبات الهوائية ومحيطها و كانت هذه الآفات متباينة الشدة بين العينات المختلفة. أظهرت نتائج اختبار الارتباط وجود علاقة ارتباط معنوية بين الاصابة بالمايكوبلازما الدجاجية الإنتانية وكل من التوسف الظهاري للقصيبات الهوائية بمستوى معنوية جيدة. لقد تم استنتاج أن الإصابات التنفسية المدروسة في الدراسة الحالية كانت فيها آفات مرضية متنوعة وكانت أكثر مزامنة وارتباطا بالإصابة بجرثومة المايكوبلازما الدجاجية الانتانية.