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STUDY ON THE INCIDENCE OF ASCITES SYNDROME IN BROILERS WITH AND WITHOUT INFECTIOUS BRONCHITIS

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ABSTRACT

The Ascites syndrome is the primary cause of death for rapidly growing broiler strains, resulting in economic loss. Avian infectious bronchitis (IB) is an acute and highly contagious respiratory disease of chickens. The disease is caused by avian infectious bronchitis virus (IBV), a coronavirus, and characterized by respiratory signs including gasping, coughing, sneezing, trachealrales, and nasal discharge.The aim of present study was to survey on the incidence of ascites syndrome in broilers with and without infectious bronchitis. In present study, 4 broiler farms were studied. Diagnosis was made based on clinical signs of IB and ascites. Then blood samples were obtained in period of 7 days for serological tests. Serological tests were ELISA and HI which were done for differentiation diagnosis with avian influenza (AI) and Newcastle disease virus. The losses were recorded with related reasons and in case of ascites, carcass was assayed accurately. Data showed that there is a direct relationship between incidence of IB and ascites syndrome.

Keywords: Ascites Syndrome, Infectious Bronchitis, Broiler Chickens

INTRODUCTION

The Ascites syndrome is the primary cause of death for rapidly growing broiler strains, resulting in economic loss **(Huchzemeyer and DeRuyck, 1986)**. Ascites is a condition that leads to accumulation of ascitic fluid, in body cavities resulting in carcass condemnation or death. Physiologically, low oxygen concentration creates an oxygen

deficit (hypoxia) and a demand for more oxygen. The increased demand may exceed the cardiopulmonary capacity to supply sufficient oxygen, resulting in pulmonary hypertension and right ventricular failure **(Julian, 1993)**.

Mortality in broiler chickens associated with fluid accumulation in the abdominal cavity is the ultimate consequence of an excessively high blood pressure in the pulmonary circulation and is known as pulmonary hypertension syndrome (PHS). The symptoms are generalized edema, hydropericardium syndrome, ascites, hypertrophy and dilatation of the heart, particularly hypertrophy of the right ventricle **(Decuypere *et al*., 2000)**.

Decreased oxygen tension or increased oxygen requirements can create hypoxic tone. Regional reductions in pulmonary oxygen tension constrict the nearby arterioles and the heart has to respond by contracting more vigorously to overcome the higher flow resistance. An increase in blood viscosity further contributes to right ventricle hypertrophy. Indeed, anoxia in birds stimulates the kidneys to produce erythropoietin which in turn stimulates the production of red blood cells in the bone marrow (erythropoiesis). This results in higher haematocrit values which are accompanied by an increase in the viscosity of the blood **(Julian, 1993)**. Together with severe pulmonary vasoconstriction; this may explain the appearance of general congestion, especially in the narrow capillaries of the lungs. The resulting hypertrophy of the right ventricle leads to a failure in the closure of the right valve between the ventricle and the atrium as a consequence, a volume of blood re-enters the atrium with each heart beat. The gradual reduction in output of the right ventricle results in a substantial increase in the venous pressure in the portal and hepatic veins, which is of special interest because the intercellular spaces in the capillary walls of the liver are larger than those in other tissues. Simultaneously, as a result of heart insufficiency, an elevated venous pressure may block the drainage capacity of the lymphatic vessels. Edema in the abdominal cavity (ascites), hydropericardium and edematous lungs are the direct results of such a change in blood pressure in different parts of the circulation **(Decuypere *et al*., 2000)**. The housing environment, including factors such as temperature (cold or fluctuating temperatures) and air quality (dust concentration, carbon dioxide levels, and oxygen levels), is known to influence the incidence of ascites in broiler chickens. The incidence of ascites greatly increases at altitudes greater than 1300 meters above sea level, presumably because of the low oxygen partial pressure **(Hernandez, 1987)**.

The metabolic rate of fast growing broiler chickens is very high and, in less well ventilated poultry house as well as at higher altitudes, oxygen becomes a limiting factor as far as their health, welfare and performance are concerned. The high metabolic demands, together with decreased availability of oxygen, may lead to hypoxia, hypoxemia and anoxia **(Julian, 1993; Maxwell *et al*., 1995; Scheel *et al*., 1992)**. The oxygen imbalance may be caused either by an extremely high metabolic demand by the tissues (resulting in anoxia, hypoxemia and hypoxia) or by an insufficient supply of oxygen (also resulting in hypoxia, hypoxemia and, finally, anoxia), or both. This imbalance is caused by exogenous as well as by endogenous (genetically based).

Avian infectious bronchitis (IB) is an acute and highly contagious respiratory disease of chickens. The disease is caused by avian infectious bronchitis virus (IBV), a coronavirus, and characterized by respiratory signs including gasping, coughing, sneezing, trachealrales, and nasal discharge. In young chickens, severe respiratory distress may occur. In layers, respiratory distress, nephritis, decrease in egg production, and loss of internal (watery egg white) and external (fragile, soft, irregular or rough shells, shell-less) egg quality are reported **(Darbyshire et al., 1979)**.

IBV was the first coronavirus described and varies greatly genetically and phenotypically, with hundreads of serotypes and strains described. Coronaviruses contain the largest known viral RNA genome in number of nucleotides, of approximately 30,000 bases. The RNA forms a single strand and single segment. IBV diversity is based on transcriptional error, which may become very relevant if occurring in genomic sequences coding for proteins, involved in adsorption to target cell or inducing immune responses. Transcriptional error variants may emerge with evolutional advantage in susceptible chickens. Large genomic changes will occur with entire gene interchanges, by reassortment, as for its replication, seven subgenomic mRNAs are produced and will enable reassortment in coinfections **(Dawson and Gough, 1971)**.

Coughing and rattling are common, most severe in young, such as broilers, and rapidly spreading in chickens confined or at proximity. Morbidity is 100% in non-vaccinated flocks. Mortality varies according to the virus strain (up to 60% in non-vaccinated flocks). Respiratory signs will subdue within two weeks. However, for some strains, a kidney infection may follow, causing mortality by toxemia. Younger chickens may die of tracheal occlusion by mucus (lower end) or by kidney failure. The

infection may prolong in the cecal tonsils **(De Witt, 2000)**.

In laying hens, there can be transient respiratory signs, but mortality may be negligible. However, egg production drops sharply. A great percentage of produced eggs are misshapen and discolored. Many laid eggs have a thin or soft shell and poor albumen (watery), and are not marketable or proper for incubation. Normally-colored eggs, indicative of normal shells for instance in brown chickens, have a normal hatchability.

Egg yield curve may never return to normal. Milder strains may allow normal production after around eight weeks **(Gelb *et al.*, 1989)**.

Chicken respiratory diseases are difficult to differentiate and may not be diagnosed based on respiratory signs and lesions. Other diseases such as mycoplasmosis by Mycoplasma gallisepticum (chronic respiratory disease), Newcastle disease by mesogenic strains of Newcastle diseases virus (APMV-1), avian metapneumovirus, infectious laryngotracheitis, avian infectious coryza in some stages may clinically resemble IB. Similar kidney lesions may be caused by different etiologies, including other viruses, such as infectious bursal disease virus (the cause of Gumboro disease) and toxins (for instance ochratoxins of Aspergillus ochraceus), and dehydration.

In laying hens, abnormal and reduced egg production are also observed in Egg Drop Syndrome 76 (EDS), cause by an Atadenovirus and avian metapneumovirus infections. At present, IB is more common and far more spread than EDS. The large genetic and phenotypic diversity of IBV have been resulting in common vaccination failures. In addition, new strains of IBV, not present in commercial vaccines, can cause the disease in IB vaccinated flocks. Attenuated vaccines will revert to virulence by consecutive passage in chickens in densely populated areas, and may reassort with field strains, generating potentially important variants.

Definitive diagnosis relies on viral isolation and characterization. For virus characterization, recent methodology using genomic amplification (PCR) and sequencing of products, will enable very precise description of strains, according to the oligonucleotide primers designed and target gene. Methods for IBV antigens detection may employ labelled antibodies, such as direct immunofluorescence or immunoperoxidase. Antibodies to IBV may be detected by indirect immunofluorescent antibody test, ELISA and Haemagglutination inhibition **(Gelb *et al.*, 2005)**. No specific treatment is available, but antibiotics can be used to prevent secondary infections. Vaccines are

available. Biosecurity protocols including adequate isolation, disinfection are important in controlling the spread of the disease. The aim of present study was to survey on the incidence of ascites syndrome in broilers with and without infectious bronchitis.

MATERIALS AND METHODS

Diagnosis and Confirmation of IB and Ascites

In present study, 4 broiler farms were studied. Diagnosis was made based on clinical signs of IB and ascites. Then blood samples were obtained in period of 7 days for serological tests. Serological tests were ELISA and HI which were done for differentiation diagnosis with avian influenza (AI) and Newcastle disease virus. The losses were recorded with related reasons and in case of ascites, carcass was assayed accurately.

Light Program

Lighting program in all farms was constant- 23 hours lightness/ 1 hour darkness.

Density

There were 10 broilers per $1m^2$.

Diet

Diet of all farms was constant and is given in **Table 1**.

Vaccination Program

Vaccination program of all farms was constant and is given as below:

Day 10: Newcastle (inj.) + AI + Clone-pronchitis (eye drop)

Day 14: Gambro (bursine-2)

Day 18: Newcastle (Avinew)

Day 21: Gambro (bursine-2)

Day 30: Newcastle (Clone)

Table 1: The Constituent Of Broilers' Diet

Age (day-old) / Constituent (Kg)	0-20	21-35	36-42	>43
Corn	555	590	630	670
Soybean	370	330	290	250
Concentrate	50	50	50	50
Oyster	15	15	15	15
Oil	10	16	16	16
Kelinacocs	0.2	0.2	0.2	0.2

RESULTS

The results of ELISA (for IB) and HI (for ND and AI) tests are given in **Tables 2-9**.

Table 2: HI test for AI and ND in farm 1

		20 day-old		27 day-old		35 day-old	
		AI	ND	AI	ND	AI	ND
Salon titr	Log 2 titr	1	1	1	1	1	1
0	0	2	0	0	0	0	0
1	1:2	3	3	2	2	0	0
2	1:4	2	2	2	1	3	0
3	1:8	1	2	3	3	2	3

4	**1:16**	**2**	**3**	**2**	**2**	**2**	**3**
5	**1:32**	**0**	**0**	**1**	**2**	**2**	**3**
6	**1:64**	**0**	**0**	**0**	**0**	**1**	**1**
7	**1:128**	**0**	**0**	**0**	**0**	**0**	**0**
8	**1:256**	**0**	**0**	**0**	**0**	**0**	**0**
9	**1:512**	**0**	**0**	**0**	**0**	**0**	**0**
10	**1:1024**	**0**	**0**	**0**	**0**	**0**	**0**
11	**1:2048**	**0**	**0**	**0**	**0**	**0**	**0**
12	**1:4096**	**0**	**0**	**0**	**0**	**0**	**0**
Min		**0**	**1**	**1**	**1**	**2**	**3**
Max		**4**	**4**	**5**	**5**	**6**	**6**
Mean		**1.8**	**2.5**	**2.8**	**3.1**	**3.6**	**4.2**

Table 3: HI test for AI and ND in farm 2

		25 day-old		**32 day-old**		**39 day-old**	
		AI	**ND**	**AI**	**ND**	**AI**	**ND**
Salon titr	**Log 2 titr**	**1**	**1**	**1**	**1**	**1**	**1**
0	**0**	**2**	**3**	**0**	**0**	**0**	**0**
1	**1:2**	**2**	**1**	**1**	**2**	**0**	**0**
2	**1:4**	**1**	**1**	**2**	**3**	**3**	**0**
3	**1:8**	**3**	**4**	**3**	**2**	**2**	**2**
4	**1:16**	**2**	**1**	**4**	**2**	**2**	**3**
5	**1:32**	**0**	**0**	**0**	**1**	**3**	**2**
6	**1:64**	**0**	**0**	**0**	**0**	**0**	**3**
7	**1:128**	**0**	**0**	**0**	**0**	**0**	**0**
8	**1:256**	**0**	**0**	**0**	**0**	**0**	**0**
9	**1:512**	**0**	**0**	**0**	**0**	**0**	**0**
10	**1:1024**	**0**	**0**	**0**	**0**	**0**	**0**
11	**1:2048**	**0**	**0**	**0**	**0**	**0**	**0**
12	**1:4096**	**0**	**0**	**0**	**0**	**0**	**0**
Min		**0**	**0**	**1**	**1**	**2**	**3**
Max		**4**	**4**	**4**	**5**	**5**	**6**
Mean		**2.1**	**1.9**	**3**	**2.7**	**3.5**	**4.6**

Table 4: HI test for AI and ND in farm 3

		23 day-old		**30 day-old**		**37 day-old**	
		AI	**ND**	**AI**	**ND**	**AI**	**ND**
Salon titr	**Log 2 titr**	**1**	**1**	**1**	**1**	**1**	**1**
0	**0**	**2**	**1**	**1**	**0**	**0**	**0**
1	**1:2**	**3**	**3**	**1**	**2**	**0**	**0**
2	**1:4**	**3**	**2**	**3**	**1**	**2**	**0**
3	**1:8**	**2**	**2**	**2**	**2**	**2**	**2**
4	**1:16**	**0**	**2**	**2**	**2**	**2**	**1**
5	**1:32**	**0**	**0**	**1**	**3**	**2**	**2**
6	**1:64**	**0**	**0**	**0**	**0**	**2**	**5**
7	**1:128**	**0**	**0**	**0**	**0**	**0**	**0**
8	**1:256**	**0**	**0**	**0**	**0**	**0**	**0**
9	**1:512**	**0**	**0**	**0**	**0**	**0**	**0**
10	**1:1024**	**0**	**0**	**0**	**0**	**0**	**0**
11	**1:2048**	**0**	**0**	**0**	**0**	**0**	**0**
12	**1:4096**	**0**	**0**	**0**	**0**	**0**	**0**
Min		**0**	**0**	**0**	**1**	**2**	**3**
Max		**3**	**4**	**5**	**5**	**6**	**6**
Mean		**1.5**	**2.1**	**2.6**	**3.3**	**4**	**5**

Table 5: HI test for AI and ND in farm 4

		27 day-old		34 day-old		41 day-old	
		AI	ND	AI	ND	AI	ND
Salon titr	Log 2 titr	1	1	1	1	1	1
0	0	0	1	0	0	0	0
1	1:2	3	4	2	3	0	0
2	1:4	3	1	3	1	2	1
3	1:8	4	2	2	1	2	2
4	1:16	0	2	2	4	2	3
5	1:32	0	0	1	1	1	1
6	1:64	0	0	0	0	3	3
7	1:128	0	0	0	0	0	0
8	1:256	0	0	0	0	0	0
9	1:512	0	0	0	0	0	0
10	1:1024	0	0	0	0	0	0
11	1:2048	0	0	0	0	0	0
12	1:4096	0	0	0	0	0	0
Min		1	0	1	1	2	2
Max		3	4	5	5	6	6
Mean		2.1	2	2.7	2.9	4.1	4.3

The results of ELISA test for detection IBV in farms is given in **Tables 6-9**.

Table 6: result of ELISA test for detection IBV in farm 1

		20 day-old	27 day-old	35 day-old
Sample	Well	Titr	Titr	Titr
Neg	A01			
Neg	A02			
Pos	A03			
Pos	A04			
1	A05	945	1432	3393
2	A06	1039	1194	3448
3	A07	945	2577	3057
4	A08	497	1346	3134
5	A09	874	1885	3368
6	A10	745	2184	3918
7	A11	621	1449	4237
8	A12	1358	1211	3918
9	B01	709	2489	4316
10	B02	1189	2094	4103
Mean		892	1786	3689

Table 7: result of ELISA test for detection IBV in farm 2

		25 day-old	32 day-old	39 day-old
Sample	Well	Titr	Titr	Titr
Neg	A01			
Neg	A02			
Pos	A03			
Pos	A04			
1	A05	947	1332	3293
2	A06	1020	1494	3348
3	A07	940	2877	3157
4	A08	490	1716	3234
5	A09	880	1715	3638
6	A10	740	2224	3848

7	**A11**	**621**	**1139**	**4307**
8	**A12**	**1348**	**1121**	**3870**
9	**B01**	**720**	**2829**	**4320**
10	**B02**	**1149**	**2174**	**4000**
Mean		**886**	**1862**	**3702**

Table 8: result of ELISA test for detection IBV in farm 3

		23 day-old	**30 day-old**	**37 day-old**
Sample	**Well**	**Titr**	**Titr**	**Titr**
Neg	**A01**			
Neg	**A02**			
Pos	**A03**			
Pos	**A04**			
1	**A05**	**890**	**1330**	**3200**
2	**A06**	**1007**	**1400**	**3400**
3	**A07**	**934**	**2750**	**3270**
4	**A08**	**470**	**1740**	**3210**
5	**A09**	**820**	**1720**	**3150**
6	**A10**	**715**	**2300**	**4002**
7	**A11**	**634**	**1240**	**4036**
8	**A12**	**1350**	**1090**	**4000**
9	**B01**	**700**	**3040**	**4200**
10	**B02**	**1170**	**2004**	**4350**
Mean		**869**	**1861**	**3682**

Table 9: result of ELISA test for detection IBV in farm 4

		27 day-old	**34 day-old**	**41 day-old**
Sample	**Well**	**Titr**	**Titr**	**Titr**
Neg	**A01**			
Neg	**A02**			
Pos	**A03**			
Pos	**A04**			
1	**A05**	**740**	**1280**	**3150**
2	**A06**	**1230**	**1460**	**3440**
3	**A07**	**920**	**2340**	**3280**
4	**A08**	**460**	**1860**	**3290**
5	**A09**	**840**	**1840**	**3140**
6	**A10**	**716**	**2340**	**4040**
7	**A11**	**624**	**1230**	**4030**
8	**A12**	**1520**	**1082**	**4090**
9	**B01**	**704**	**3024**	**4250**
10	**B02**	**1700**	**2044**	**4380**
Mean		**945**	**1850**	**3709**

DISCUSSION

The objective of the present study was on the incidence of ascites syndrome in broilers with and without infectious bronchitis. In the present study, we only studied seven poultry houses which showed ascites syndrome. **McGovern *et al.*, (2001)** reported that the birds in the low CO_2 treatment did not have a reduced ascitic score compared to the birds in the raised high CO_2 treatment, however, the right ventricle area was significantly reduced in

the low CO_2 treatment from 0.50 to 0.47 cm^3 suggesting more ascites syndrome percent in the high CO_2 treatment and under recommended management practices in Alberta, CO_2 and O_2 levels should not be a contributing factor to the incidence of ascites. Some believes that ascites may be a consequence of lowered oxygen tension in poultry sheds caused by increased quantities of dust or noxious fumes as a result of poor ventilation **(Pattison, 1993)**. It is clear from this study that there is a relation between the incidence of ascites syndrome and the ventilation factor in broiler chickens. In this study, we could reduce nearly the incidence rate of ascites syndrome by 1 percent, and the prevalence of CRD complex reduced from 7 percent to 3 percent in seven poultry houses, and the food conversion ratio (FCR) changed. Ascite is one of the non-infective poultry diseases in world **(Calnek et al., 1991)**. In our country because of inappropriate and bad management conditions its prevalence is very high and losses due it reported in high levels. According to obtained data from 18 countries of 4 continents revealed that this syndrome loss is about 4.7% **(Hassanzadeh, 2009)**. This syndrome is multi-reason and of its origins can be refer to endogen and exogen agents **(Guo et al., 2007)**. Of its important causes can be referring to imbalance between required and existed oxygen. Oxygen deficiency consequent several factors such as low temperature, existence of Cartilaginous and bony nodules in lungs and pulmonary disease such as bronchitis, mycoplasmosis, colibacillosis and aspergillosis, rapid growth, height, poisoning, inappropriate ventilation **(Decuyper *et al.*, 2000)**. However, this syndrome can be appear after disease such as CRD complex and infective bronchitis as a result of complication **(Calnek *et al.*, 1991)**. In general, ascitic losses in broilers are as a result of pulmonary hypertension that results into tachycardia and heart hypertrophy and finally ascite. Pellets feed increased intake of energy per unit time, which results in increased requests for oxygen to high metabolism and high density is caused hypoxia **(Dale and Villacres, 1986)**. Rechelmann showed that peppermint extraction has anti cough and nasal decongestant effects which are causes to decrease in surface tension of synthetic surfactant that can reduce pulmonary surface tension. Also demonstrated that use of peppermint essential oils result in destruction of airways discharges. Also demonstrated that use of peppermint essential oils result in decreasing of mucosal hypertrophy, losses of goblet cell and mucosal accumulations in trachea and minimizing the neutrophils infiltration. All

above mentioned remarks causes increase in animal protectivity against secondary respiratory infections **(Elie *et al.*, 2006)**. According to other researches, peppermint extraction has antimicrobial effects against *E.coli, Clostridium perfringens, lactobacillus bulgaricus,* and *Streptococcus thermophilus*.

CONCLUSION

From this study, the following conclusions can be drawn:

This study confirms that carbon dioxide and oxygen contribute significantly to the incidence of ascites under unsuitable ventilation, and it is possible to decrease the incidence rate of ascites by correction of the poultry house conditions.

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