RETROSPECTIVE ANALYSIS OF THE FREQUENCY AND CONTRIBUTORY CAUSES OF ASCITES SYNDROME IN BROILERS IN SOUTH BANAT

Pavle Gavrilović, Aleksandar Živulj, Igor Todorović

Veterinary Specialized Institute “Pančevo”, Novoseljanski put 33, 26000 Pančevo, Serbia
Corresponding author: Pavle Gavrilović, e-mail address pavlelula@yahoo.com
Original scientific paper

Abstract: Ascites syndrome is a multi-factorial, noncontagious disease of broilers. Chickens selected for rapid growth have a high basal metabolism and consequently increased demands for oxygen. Poor environmental conditions and other unfavourable factors which reduce the amount of available oxygen cause hypoxia, pulmonary hypertension, right ventricular dilation and right heart failure, which results in generalized passive hyperaemia of organs and ascites as the most striking gross pathology manifestation. The aim of the study was to investigate, retrospectively, the frequency of the disease in a selected district with widespread poultry production and to identify main factors that contribute to the outbreak of the disease. In the period from 2011 to 2017 ascites syndrome was diagnosed by post mortem examination of chickens in 12 out of 91 flocks with health disorders, examined in the Veterinary Specialized Institute "Pančevo". Based on anamnesis, signs of disease and pathomorphological findings, poor environmental conditions and inadequate feed were identified as main contributory factors. In the majority of cases ascites syndrome occurred in small flocks, raised in unsuitable environmental conditions.

Key words: ascites syndrome, chickens, broilers, environmental conditions

Introduction

Ascites syndrome is one of the diseases that continuously affect poultry industry in South Banat over the past decade. The disease is also known as pulmonary hypertension syndrome, waterbelly, right ventricular failure and under some other descriptive terms which do not indicate the aetiology of the disease (Palić et al., 1994; Knežević and Matejić, 1996). Ascites syndrome is a multi-factorial, noncontagious disease of broilers, described for the first time in broilers...
raised at high altitudes in Bolivia (Hall and Machicao, 1968). It is estimated that 4.7% of the broilers worldwide have the disease (Maxwell and Robertson, 1997).

The latest generation of hybrids of domestic hen (Gallus gallus domesticus) have been selected for a more rapid growth and more intensive protein synthesis which requires more oxygen (Decuypere et al., 2005). However, the capacity of the cardiovascular system, which the selection could not significantly influence, has its own physiological limits (Lorenzoni et al., 2006) and cannot always respond to increased oxygen demands necessary for rapid growth. Hypoxia triggers a series of events in the organism that result in the development of a metabolic disorder, characterized by hypoxaemia, increased workload of the cardiopulmonary system and central venous congestion (Baghbanzadeh and Decuypere, 2008). The most characteristic gross pathology lesions are ascites, dilation and hypertrophy of the right ventricle (Knežević and Matejić, 1996).

Fluid exchange across the walls of capillaries, according to Starling law, is regulated by the physiological values of hydrostatic and colloidal osmotic pressures in and out the blood vessels. Changes in these values, that affect the mechanism of tissue fluid exchange, lead to oedema which can occur due to increased intravascular hydrostatic pressure, decreased plasma colloidal osmotic pressure, increased vascular permeability, obstruction of lymph drainage and renal retention of salt and water (Knežević and Jovanović, 1999). Increased intravascular hydrostatic pressure can be caused by hepatic and cardiac diseases, and pulmonary hypertension (Currie, 1999). Since plasma proteins, in particular albumin, are responsible for colloidal osmotic pressure, pathological conditions with reduced synthesis or loss of albumin can cause ascites (Baghbanzadeh and Decuypere, 2008). Increased vascular permeability can be caused by various chemical compounds such as phenol and dioxin derivatives (Balog, 2003). According to the literature, primary and contributory causes of ascites include: high altitude, rapid growth rate, pulmonary disease, high energy ration, pelleted feed, cold, the presence of harmful gases and dust particles in the air, high salt concentration in feed, phosphorus deficiency, hepatotoxins, mycotoxins, furazolidone, Se and vitamin E deficiency, stress etc. (Lister, 1997).

Due to the lack of data related to ascites syndrome in broilers in the Republic of Serbia, we decided to investigate the frequency of the disease in a selected district with widespread poultry production and to identify main factors that contribute to the outbreak of the disease.

Material and Methods

Material for examination consisted of 595 corpses of broilers which had been delivered to the laboratory of the Veterinary Specialized Institute “Pančevo” during seven consecutive years from 2011 to 2017. The samples originated from
91 flocks with manifested health disorders from South Banat district. On the receipt of the samples detailed anamnesis was taken, including questions about the flock size, manifestation of signs of disease, morbidity and mortality rate, environmental conditions and feed mixtures used. All delivered samples were necropsied and examined pathomorphologically according to the official procedure described by Marinković and Nešić (2013).

**Results and discussion**

The necropsy revealed gross lesions typical of ascites syndrome in broilers from 12 flocks, which is 13.19% of all the examined flocks (Table 1). The lesions appeared in broilers from 3 to 6 weeks (Figure 1). The course of the disease was acute in 3 flocks and subacute in 9 flocks. Mortality ranged from 4.6 to 60.0% (Table 2). Based on anamnesis, signs of disease and gross pathology findings, diagnosed cases of ascites syndrome were classified in three groups.

**Table 1. The frequency of ascites syndrome in broiler flocks examined in VSI “Pančevo”**

| Year | Examined flocks (N) | Flocks with ascites syndrome (N) | Flocks with ascites syndrome (%) |
|------|---------------------|---------------------------------|---------------------------------|
| 2011 | 17                  | 2                               | 11.76                           |
| 2012 | 15                  | 2                               | 13.33                           |
| 2013 | 20                  | 3                               | 15.00                           |
| 2014 | 16                  | 1                               | 6.25                            |
| 2015 | 6                   | 1                               | 16.67                           |
| 2016 | 2                   | 1                               | 50.00                           |
| 2017 | 15                  | 2                               | 13.33                           |
| Σ    | 91                  | 12                              | 13.19                           |

**Table 2. Age of chickens in which ascites syndrome was diagnosed, course of the disease and mortality rate**

| Flock | Year | Age (weeks) | Course | Mortality (%) |
|-------|------|-------------|--------|---------------|
| 1     | 2011 | 5           | acute  | 6.25          |
| 2     | 2011 | 6           | subacute | 4.60         |
| 3     | 2012 | 3           | subacute | 45.45       |
| 4     | 2012 | 4           | subacute | 60.00       |
| 5     | 2013 | 5           | acute  | 18.00         |
| 6     | 2013 | 4           | acute  | 15.00         |
| 7     | 2013 | 6           | subacute | 20.20      |
| 8     | 2014 | 6           | subacute | 30.00       |
| 9     | 2015 | 5           | subacute | 20.10       |
| 10    | 2016 | 4           | subacute | 5.00        |
| 11    | 2017 | 5           | subacute | 20.00       |
| 12    | 2017 | 3           | subacute | 30.00       |
Figure 1. Gross pathology findings in broilers affected with ascites syndrome: (a) Swollen abdomen; (b, c) Accumulation of fluid in pleuropertitoneal cavity; (d) Passive hyperaemia and degenerative necrotic changes of the liver, ascites and hydropericardium; (e, f) right ventricular dilation. (Photo: P. Gavrilović)

Group 1 (flocks 1, 5, 6, 7, 8, 9, 10, 11 and 12)

Chickens from this group originated from small individual holdings and were fed pelleted feeds. Signs of disease included slower movement, blue
discolouration of the comb and wattles (cyanosis), abdominal distension and dyspnoea. The gross pathology findings were characterized by ascites, hidropericardium, right ventricular dilation, hyperaemia of the lungs, liver, spleen, kidneys and intestine. In the facilities in which these 9 flocks were raised, the zoosanitary regimes were not adequate. Low ambient temperature, inadequate ventilation and sawdust bedding with a lot of dust were identified as potential contributory causes of ascites syndrome in this group. The experimental studies have shown positive correlation between ascites incidence and low ambient temperatures. Cold temperatures influence the occurrence of ascites by increasing metabolic oxygen requirements and consequent pulmonary hypertension (Julian et al., 1989; Stolz et al., 1993). Poor air quality, dust and respiratory diseases can cause respiratory damage and predispose birds to ascites syndrome. Ammonia, carbon monoxide, carbon dioxide, dust and humidity are recognized air contaminants that can increase susceptibility to ascites syndrome (Afolayan et al., 2016). In addition to the above environmental conditions, feeding regime could have an impact on the outbreak of the disease. Hasani et al. (2018) showed that in broilers the occurrence of ascites syndrome under mash-fed regime was less than in pellet- and crumble-fed groups.

Group 2 (flock 2)

Chickens from flock 2 originated from a farm with high level of zoosanitary and biosecurity measures. Signs of disease were manifested as reduced growth and poor feathering. Chickens did not respond to antibiotic therapy. The necropsy revealed ascites, hepatic necrosis, gizzard erosions and enteritis. Reduced growth and poor feathering in association with gizzard erosions, enteritis and hepatic necrosis indicate inadequate feed as a potential cause of the disease in this flock. Nutrient factors exhibit their effects by different mechanisms and they can have synergistic effects. The presence of hepatotoxins in feed can cause the liver damage and ascites (Firestone, 1973). Excess in sodium causes the increase in blood pressure, while vitamin E deficiency, for example, predisposes tissue damage caused by free radicals and leads to degenerative changes in the myocardium (Julian, 1987; Aftab and Khan, 2005).

Group 3 (flock 3 and 4)

Broilers from flocks 3 and 4, in which mortality was 45.45% and 60.0%, exhibited first signs in the second week of life in the form of digestive system disorders manifested as diarrhea. The mortality was constantly increased until the third week when a sudden peak occurred. Gross pathology lesions included ascites, hydropericardium, right ventricular dilation, hyperaemia of the lungs, liver, spleen, kidneys, degenerative necrotic changes in the liver, gizzard erosions and enteritis.
Beside the same clinical manifestations and gross pathology findings, it was common for the chickens from these two flocks that they were fed complete feed mixture of a same manufacturer. Based on anamnesis, signs of disease and gross pathology findings, it was suspected intoxication as a cause of health disorders in these two flocks. Similar gross lesions were described by Ivetić et al. (2003) in broilers fed feed mixtures that contained poor quality fats. The literature describes intoxication in chickens accompanied with ascites due to toxic components in certain feed fats such as derivatives of dibenzo-p-dioxin (Firestone, 1973).

**Conclusion**

In broilers investigated within this retrospective study, ascites syndrome mostly appeared in small flocks raised on smallholdings under unsuitable zoosanitary regimes. The main factors contributing to the incidence of the disease were inadequate feed and environmental factors, primarily low ambient temperature and poor ventilation.

**Retrospektivna analiza učestalosti i uzroka koji doprinose pojavi ascites sindroma kod brojlera u južnom Banatu**

*Pavle Gavrilović, Aleksandar Živulj, Igor Todorović*

**Rezime**

Ascites sindrom je nekontagiozno oboljenje brojlera multifaktorijalne etiologije. Pilići selekcionisani na brz rast imaju visok bazalni metabolizam zbog čega su povećane potrebe organizma za kiseonikom. U nepovoljnim ambijentalnim uslovima kao i pod dejstvom drugih štetnih uticaja koji smanjuju količinu raspoloživog kiseonika dolazi do hipoksije, plućne hipertenzije, dilatacije i insufficijencije desnog srca koja ima za posledicu generalizovanu pasivnu hiperemia organa i ascites kao najupečatljiviji patoanatomski nalaz. Cilj studije bio je da se retrospektivno istraže učestalost i glavni činioci koji doprinose pojavi oboljenja na odabranom području sa rasprostranjenu živinarskom proizvodnjom. U periodu od 2011. do 2017. godine ascites sindrom je dijagnostikovan patoanatomskim ispitivanjem kod pilica iz 12 od 91 jata sa zdravstvenim problemima ispitivanim u Veterinarskom specijalističkom institutu „Pančevo“. Na osnovu anamneze, kliničke slike i patoanatomskog nalaza kao glavni činioci koji doprinose pojavi oboljenja identifikovani su nepovoljni ambijentalni uslovi i
neadekvatna hrana. U najvećem broju slučajeva ascites sindrom se javljao u malim jatima, gajenim u neadekvatnim ambijentalnim uslovima.

Ključne reči: ascites sindrom, pilići, brojleri, ambijentalni uslovi

References

AFOLAYAN M., ABEKE F. O., ATANDA A. (2016): Ascites versus sudden death syndrome (SDS) in broiler chickens: a review. Journal of Animal Production Research, 28, 2, 76-87.
AFTAB U., KHAN A. A. (2005): Strategies to alleviate the incidence of ascites in broilers: a review. Brazilian Journal of Poultry Science, 7, 4, 199-204.
BAGHBANZADEH A., DECUYPERE E. (2008): Ascites syndrome in broilers: physiological and nutritional perspectives. Avian Pathology, 37, 2, 117-126.
BALOG J. M. (2003): Ascites syndrome (pulmonary hypertension syndrome) in broiler chickens: are we seeing the light at the end of the tunnel? Avian and Poultry Biology Reviews, 14, 3, 99-126.
CURRIE R. J. W. (1999): Ascites in poultry: recent investigations. Avian Pathology, 28, 4, 313-326.
DECUYPERE E., HASSANZADEH M., BUYS N. (2005): Further insights into the susceptibility of broilers to ascites. The Veterinary Journal, 169, 3, 319-320.
FIRESTONE D. (1973): Etiology of chick edema disease. Environmental health perspectives, 5, 59-66.
HALL S. A., MACHICAO N. (1968): Myocarditis in broiler chickens reared at high altitude. Avian Diseases, 1, 75-84.
HASANI A., BOUYEH M., RAHATI M., SEIDAVI A., MAKOVICKY P., LAUDADIO V., TUFARELLI V. (2018): Which is the best alternative for ascites syndrome prevention in broiler chickens? Effect of feed form and rearing temperature conditions. Journal of Applied Animal Research, 46, 1, 392-396.
IVETIĆ V., VALTER D., PAVLOVIĆ I., MILJKOVIĆ B., MASLIĆ-STRIŽAK D., ILIĆ Ž., SAVIĆ B., STANOJEVIĆ S., SPALEVIĆ LJ. (2003): Atlas bolesti živine. Naučni institut za veterinarstvo Srbije, Beograd. pp 156.
JULIAN R. J. (1989): Lung volume of meat-type chickens. Avian Diseases, 33, 1, 174-176.
JULIAN R. J. (1987): The effect of increased sodium in the drinking water on right ventricular hypertrophy, right ventricular failure and ascites in broiler chickens. Avian Pathology, 16, 61–71.
KNEŽEVIĆ M., JOVANOVIĆ M. (1999): Opšta patologija. Makarije, Beograd, pp 306.
KNEŽEVIĆ N., MATEJIĆ M. (1996): Bolesti pernate živine. Veterinarski fakultet, Beograd, pp 185.
LORENZONI A. G., RUIZ-FERIA C. A. (2006): Effects of vitamin E and L-arginine on cardiopulmonary function and acites parameters in broiler chickens reared under subnormal temperatures. Poultry Science, 85, 12, 2241-2250.
LISTER S. (1997): Broiler ascites: a veterinary viewpoint. World's Poultry Science Journal, 53,1, 65-67.
MARINKOVIĆ D., NEŠIĆ V. (2013): Tehnika obdukcije životinja sa osnovama tanatologije. Fakultet veterinarske medicine, Univerziteta u Beogradu, Beograd, pp 177.
MAXWELL M. H., ROBERTSON G. W. (1997): World broiler ascites survey 1996. Poultry International, 36, 4, 16-30.
STOLZ, J. L. (1993): Ascites syndrome mortality and cardiological response of broiler chickens subjected to cold exposure: effects of verapamil and L-carnitine supplementation. Ph.D. Dissertation. Texas A&M University.
PALIĆ T., RAJIĆ I., NIKOLIĆ Z. (1994): Bolesti živine, Poremećaji ishrane. Savez živinara Srbije, Beograd, pp 123.

Received 22 May 2019; accepted for publication 17 June 2019