**UNUSUAL PATHOMORPHOLOGICAL FINDINGS IN ONE-DAY-OLD POULTRY: CASE REPORTS**

Marina Tišljar1\*, Tajana Amšel Zelenika1, Vladimir Savić1, Borka Šimpraga1, Fani Krstulović1, Tihomir Zglavnik1

1Croatian Veterinary Institute, Poultry Centre; Zagreb, Croatia

\*Marina Tišljar, DVM, PhD; Croatian Veterinary Institute, Poultry Centre, Heinzelova 55, 10000 Zagreb; tel: +385 (01) 2440 214; fax: +385 (01) 2441 396; e-mail: tisljar@veinst.hr

**Summary**

### Background: Two cases of gross lessions closely resembling runting stunting syndrome (RSS) / malabsorption syndrome (MAS) in 1-day-old pullets (case 1) and in poults (case 2),were described in the present paper. Furthermore, along with these extremely rare pathomorphological changes in 1-day-old poultry, haemorrhagic bursitis in pullets and signs of generalised arteriosclerosis in poults were histopathologically confirmed. A review of the available literature data did not yield any report on at least one of the pathomorphologically confirmed conditions occurring naturally in 1-day-old poultry, as presented in this case reports. The owners’ limited anamnestic information (e.g. about the vaccination in both young poultry and in the parent flocks), and their permission for only a few additional examination procedures in both cases, left us in the position of speculating on the possibility of vertical transmission of RSS (both cases); infectious bursal disease virus (IBDV) breakthrough infection (case 1), and hatchery technology or genetic impact in case of generalised arteriosclerosis in day-old poults (case 2).

**Case presentations**: A few months ago, an increased mortality rate was observed in a pullet flock aged up to 2 weeks at one Croatian farm (case 1). Gross lesions, closely resembling RSS, were seen in all (52) the examined pullets aged 1, 4 and 15 days. In 30 (57.69 %) carcasses of 1- to 4-day-old pullets, haemorrhagic bursitis was also sporadically observed. During a routine necropsy in 2014 and 2015, in 50 (65.79 %) out of a total of 76 male broiler turkey carcasses coming from different hybrid lines, pathomorphological changes typical of RSS / MAS (notably degenerative changes in the pancreas) were confirmed in newly hatched poults (case 2). In 0-day-old and in the poults aged two weeks, various generalised arteriosclerotic changes were histopathologically confirmed in all the examined organs. In both cases, the predominant lesions of RSS / MAS were also confirmed at the age of 15 days. As an accompanying finding, *Escherichia coli* (*E. coli*) was confirmed in all the pullet age groups, and turkey astrovirus 1 (TastV-1) was isolated from only one poult aged 15 days.

**Conclusions:** The pathomorphologically diagnosed conditions are important because they are described and reported for the first time in the presented age groups of the poultry (0, 1 and 4 days of age). In the authors’ opinion, these reports on potentially immunosuppressive conditions and diseases in 0- and 1-day-old poultry should stimulate further research in the identification and erradication of key etiopathogenetic factors.

**Key words:** pathomorphology; pullet; poult; haemorrhagic bursitis; infectious bursal disease (IBD); runting and stunting syndrome (RSS); malabsorption syndrome (MAS); colibacillosis; arteriosclerosis

**Introduction**

Two cases of pathomorphological findings closely resembling RSS, were diagnosed in newly hatched and 1- to 15-day-old poults and pullets, respectively (Laboratory of Pathology, Croatian Veterinary Institute, Poultry Centre, Zagreb). More unusual was the finding of haemorrhagic bursitis in 1- and 4-day-old pullets as, according to the basic anamnestic data, they had already been protected against IBDV infection by maternal antibodies. In the poults aged 0 day to 2 weeks, histopathology confirmed generalised arteriosclerosis – another and not less unusual finding in this poult age group. The pullets were also infected with *E. coli*.

Runting and stunting syndrome (RSS) or malabsorption syndrome (MAS) in chickens and turkeys is considered a transmissible disease of uncertain aetiology (Kang *et al.,* 2012). According to Bracewell and Randall (1984), the field cases of RSS in broilers and turkeys are detectable as early as at the age of 4 days. Bacterial, protozoal and viral agent contributions in the development of RSS outbreaks depend on flock management and flock immunological status (Nuñez & Ferreira 2013, Moura-Alvarez *et al.,.* 2013, Moura-Alvarez *et al.,* 2014). Among the viral agents, enteric viruses, such as chicken astrovirus (CAstV) and turkey astroviruses 1 and 2 (TAstV 1 and 2), chicken parvovirus (ChPV), avian rotavirus (ART), fowl adenovirus type I (FAdV I), and avian reovirus (AReo) have been confirmed as the most important factors in RSS etiopathogenesis. Gross lessions usually include enlarged intestine with fluids, inflamed-like proventriculi, pancreas degeneration, and soiled vents (Guy, 1998; Zavala and Sellers, 2005; Zavala, 2006). Chicks affected with RSS often exhibit degenerative lesions of exocrine pancreas (atrophy, fibroplasia, vacuolation and occasional necrosis in acinar cells) (Bracewell and Randall, 1984; Kouwenhoven *et al.,* 1986; Qamar *et al.* 2013). In turkeys, these changes can be seen as early as on day 8 of age (Summers, 2008). Apart from clinical symptoms of chicken uneven growth in field cases of RSS detectable as early as on day 4 of age (usually between 1 and 2 weeks of age) (Kouwenhoven *et al.,* 1978; Summers, 2008), until now, there were no literature data about gross lesions resembling RSS in 0- and 1-day-old pullets and/or poults.

Regarding poultry immunological status, acute highly contagious infectious bursal disease (IBD) (Rauf *et al.,* 2011) caused by a  serotype 1 infectious bursal disease virus (IBDV) (genus *Avibirnavirus,* family *Birnaviridiae*)  (Bolis *et al.,* 2003), is one of the most immunosuppressive diseases. In the IBD acute form (affected are mainly chickens aged 3-6 weeks), the typical gross lesions are characterised by enlarged and swollen bursa of Fabricus (BF) with haemorrhages (Lukert and Saif, 2003; Singh *et al.,* 2015). In the first few weeks of life, chickens are usually protected from IBDV by the maternal antibodies, and thereafter by the antibodies produced by vaccination with live attenuated IBD vaccines (Muskett *et al.,* 1979; Wyeth *et al.,* 1981). While some virus strains may cause up to a 60-% mortality in chickens aged 3 weeks and older, the other symptoms are related to a strong, prolonged immunosuppression without visible clinical symptoms in chickens infected at an early age (the so-called *silent infections*; Eterradossi and Saif, 2013). According to them, in chicks with maternal antibodies, the infection is usually confirmed at necropsy by macroscopically and microscopically found bursal atrophy. So far, there have been no reports on infections followed by haemorrhagic bursitis in 1-day-old chickens protected by maternal antibodies. This also includes the field case of possible idiopathic haemorrhagic bursitis or the occurrence of haemorrhagic bursitis caused by any other sort of microorganisms in newly hatched chicks.

In neonatal chicks, colibacillosis, a syndrome caused by *E. coli,* may appear if they are of poor quality and/or because of inappropriate sanitation in the hatchery leading to early chick death. According to the classification of infections and syndromes caused by avian pathogenic *E. coli* (APEC) (Nolan *et al.,* 2013), the neonatal chickens usually suffered from localised forms - coliform omphalitis / yolk sac infection and/or coliform cellulitis (inflammatory process) as well as from colisepticaemia (systemic infection). In most field cases, *E. coli* often causes a systemic infection concurrent with other diseases or manifests itself after the bird has suffered from infectious (IBD, chicken infectious anaemia /CIA/, infectious bronchitis /IB/, etc.), poisoning, nutritional and/or traumatic diseases. So far, there have been no reports on field cases of haemorrhagic bursitis in 1-day-old chickens infected with *E.* *coli,* IBDV, chicken infectious anaemia virus (CIAV), Marek’s disease virus (MDV); chicken astrovirus (CAstV), rotavirus, reovirus, chicken parvovirus (ChPV), fowl adenovirus of subgroup I (FAdV-1), or avian nephritis virus (ANV). The most frequent bursal pathological change induced by all these microrganisms (except by IBDV) is a marked lymphocyte depletion of the bursa of Fabricius in chickens older than 1 or 4 days (Nakamura *et al.*, 1985; Nakamura *et al.*, 1990; Reynolds and Schultz-Cherry,2003)**.**

Arteriosclerosis (a generic term for arterial wall thickening and loss of elasticity /„hardening of arteries“/; Mitchell, 2015) is common in many different species of birds (Julian, 2002). It is a regular finding in male turkey broilers died of perirenal haemorrhages (8th – 19th week of life), and Julian (1996) considered it a posible result of hypertension. Since broiler turkeys aged 8 – 19 weeks belong to the category of domestic poultry suffering from acute cardiovascular diseases (CVD) followed by a high mortality, the findings of such drastic degenerative changes in the arteries of the youngest examined pullets may represent a real additional threat to the function of cardiovascular system over the whole period of fattening.

In both cases, the number of additional investigations was extremely limited due to the owners’ decisions. Therefore, it could only be speculated about the possible causes of these unusual patomorphological findings in 0-day and 1-day-old poultry (typical of RSS and haemorrhagic bursitis in the pullet, and RSS and generalized arteriosclerosis in chickens) and the effect of interconnecting etiopathogenic factors on health status during the production period could only be guessed on the basis of literature data.

**Material and Methods**

**Cases description and sampling methods**

***Case 1***

In 2016, the mortality in investigated pullet flock was increased in the first two weeks of age. According to basic anamnestic data, 807 (1.66 %) out of the total 48, 654 chicks imported from one of the EU countries, died. During the first week 595 (1.22 %) died, and in the second week 212 (0.43 %) chicks were lost. Upon arrival, the chicks were vaccinated against coccidiosis (PARACOX R, Intervet), and at the age of 10 days they received the vaccine against salmonellosis (Salmonella Vac E, Lohmann). Fifty-two chicks (1, 4 and 15 days of age) were necropsied at the Laboratory of Pathology, Croatian Veterinary Institute, Poultry Centre, Zagreb. The organ samples for aerobic bacteriology (liver, spleen, intestine, yolk sac, heart samples) and histopathology (liver, lymphoid organs /bursa of Fabricius, thymus, spleen/, duodenum, jejunum, pancreas, brain, heart, glandular stomach, kidney samples) examinations were applied according to the owner's request. In 30 (57.69 %) 1- to 4-day-old chicks, gross lesions closely resembling RSS (the poult size nonuniformity, pancreatic fibrosis, proventricular hyperplasia, catarrhal enteritis, and ochre yellow, foamy content in caeca) were seen in all cadavers, including the sporadic findings of haemorrhagic bursitis. Histpathological changes confirmed the macroscopic findings. In all the chicks omphalitis/yolk sac infection and/or signs of sepsis were also confirmed. The most prevalent pathomorphological findings in 22 (42.30%) chicks aged 15 days were typical of RSS. Infection with *E. coli* was determined in all the pullet groups. The anamnestic data included the information about parent flock vaccination against IBDV infection, but without any detail regarding the origin of vaccine and the age at which the parent flock was vaccinated.

**Case 2**

Out of the total of 76 broiler turkey carcasses from different hybrid lines, necropsied at the Laboratory of Pathology, Poultry Centre, Croatian Veterinary Institute, Zagreb, Croatia, over the period 2014 –2015, in 50 (65.79 %) newly hatched poults, and in 26 (34.21 %) 1- to 2-week-old poults, observed were macroscopic and microscopic pathomorphological changes typical of RSS / poult malabsorption syndrome (PMAS) (notably degenerative changes in the pancreas), and sporadic omphalitis and/or yolk sac infection. The repeatedly confirmed pathomorphological findings of the poult size nonuniformity, of degenerative changes in 0-day-old poults pancreases, suggested the possibility of the vertical transmission of RSS/PMAS. Only in one case (poult aged 2 weeks), the turkey astrovirus 1 (TastV-1) was isolated. Additionally, histopathologic examination confirmed generalized arteriosclerosis in all 0-day to 2-week-old poults. Taking into account a high cardiovascular mortality rate in male turkey broilers (aortal rupture; perirenal haemorrhages and sudden death syndrome) over the period 8th – 19th week of life, common in world and domestic broiler turkey production, the finding of generalised arteriosclerotic changes in all the examined poults (with the emphasis on 0-day-old poults) seemed to be vital. The owners could not inform us about the vacciantion programme implemented in the parent flocks, and the virological and histopathological investigations were performed according to their request. The organ samples of the heart, duodenum and pancreas, the blood vessels / aorta, pulmonary artery, the liver, bursa of Fabricius, thymus, spleen, brain, kidney, and the glandular stomach, were taken for the histopathological examination, and the intestine, spleen, kidney, bursa of Fabricius and the liver were submitted to the molecular diagnostic technique for isolation of *Mycoplasma gallisepticum,* avian reovirus, turkey coronavirus, astrovirus, and chicken and turkey reovirus***.***

***Pathomorphology analysis***

The tissue samples were fixed in 10-% neutral formalin, and embedded in paraffin. Sections thick 5 μm were cut on a rotary microtome (*MICROM, Zeiss*, Austria) and stained with haematoxylin-eosin (HE). The slices were analysed under the light microscope (LEICA *DMLB*, Germany). The images were captured using the PIXERA Pro150ESdigital camera *(*Pixera Corporation, USA).

***Bacteriology analysis***

*Salmonella* spp. *isolation*

Salmonella was isolated from the organs following the instructions for the standard EN ISO 6579:2002.

*Escherichia coli isolation*

At isolating the bacterium *Escherichia coli*, all the analysed organs were trans-inoculated onto the blood agar (Columbia agar with the supplementation of 5%-10% of sheep blood), Columbia agar and MacConkey agar. Simultaneously, the organs were trans-inoculated onto the TBX agar, a selective medium for *E. coli* isolation (Barnes *et al*., 2003). Biochemical characterisation was determined using the API system ID 32E (*bioMérieux*, France).

***Molecular diagnostic testing for Mycoplasma gallisepticum, avian reovirus, turkey coronavirus, astrovirus, and chicken and turkey reovirus determination***

*Molecular diagnosis*

The polymerase chain reaction (PCR) method was used to detect *Mycoplasma gallisepticum* DNA, and the method of reverse transcription (RT) and PCR for the presence of avian reovirus, turkey coronavirus and astrovirus RNA. Total bacterial and viral DNA and RNA were obtained from homogenised organs using High Pure Viral Nucleic Acid Kit Kit (*Roche Applied Science*, Mannheim, Germany) according to the manufacturer's instructions. The presence of *M. gallispticum* DNA was searched by real-time PCR (Real Time PCR) method according to Raviv and Kleven (2009). The presence of chicken and turkey reovirus RNA, as well as the turkey coronavirus RNA, were searched using the method of Real Time RT-PCR according to Spackman *et al.* (2005), while the presence of turkey astrovirus types 1 and 2 RNA was determined by conventional RT-PCR method by Dayu *et al.* (2007).

**Results and Discussion**

A total od 52 pullets (1, 4 and 15 days of age) was investigated. In 30 (57,69%) 1- to 4-day-old pullets, gross lesions typical of RSS (poult size nonuniformity, pancreatic fibrosis, proventricular hyperplasia, catarrhal enteritis, and ochre yellow, foamy content in the caeca) were seen in all carcasses, and findings of haemorrhagic bursitis 1-day-old chicks and in two 4-day-old chicks. In all these pullets omphalitis/yolk sac infection and/or signs of sepsis were also confirmed. The most prevalent pathomorphological findings in 22 (42.30%) pullets 15 days of age were typical of RSS (case 1). Similar gross lesions in chickens suffering of RSS were described by Zavala and Sellers (2005) and Zavala (2006). In all examined pullets the histopathologic examination confirmed fibroplastic changes and focal mononuclear cell hyperplasia in the pancreas associated with the findings of epithelial and mononuclear cell hyperplasia in proventricular lamina propria; desquamative, catarrhal or haemorrhagic duodenitis and jejunitis with abundant rod-like shaped microorganisms and rare interspersed cystic enlargement crypts. There was also a focal encephalomalacia in the cerebellum. Histologic studies of pancreatic lesions in broilers with RSS revealed fibrosis, inflammation, vacuolar changes in the exocrine compartment, degeneration of acinar cells and loss of zymogen granules (Qamar *et al.,* 2013). Twenty-four hours after the experimental exposure the chicken to ANV-1, ANV-2, chicken parvovirus and a novel chicken astrovirus, the cystic lesions were present in the small intestine (Kang *et al.,* 2012). Although the RSS aetiology remains unknown, early investigations revealed a probable viral aetiology (Decaesstecker *et al.,* 1988; Smart *et al.,* 1988). An important finding was the detection of CAstV in 1-day-old breeder chicks, which may indicate a vertical transmission (Mettifogo *et al.,* 2014). Unfortunately, in spite of present rare pathomorphological changes that in all the examined day-old pullets resembled on RSS, and therefore raises suspicion of vertical transmission of the disease, the virological investigation could not be performed according to the owner's request.

The extensive haemorrhages in the bursas in examined 1- to 4-day-old pullets, with sparsely disseminated, clearly demarcated follicles populated with lymphoblasts, and a prominent hyperplastic epithelium of the folds, represented almost unique findings in farm-reared 1- and 4-day-old chicks protected with maternal antibodies against IBDV infection. Namely, in most acute cases of IBD the histopathologic findings display intra- and interfollicular haemorrhages, but not the picture of regular, oval, sparsely disseminated lymphoblasts populated follicles „immersed“ in „haemorrhagic mass“ in the bursa of 1- and 4-day-old chicks. The microscopic picture of extensive interstitial haemorrhages and the prominent oval foliclles filled with lymphoblasts (but without intrafollicular bleeding) were not typical findings of even acute IBDV infection. **T**he lymphoblastic transformation in chicken embryo bursa begins when the nodule reaches approximately 60 µm in diameter, and give rise to a developmental lymphocytes series (Ackerman and Knouff, 1964). In the follicles of definitive bursa of Fabricius the lymphoblasts can be seen, but the most prevalent are the lymphocyte cells (Thorbecke *et al.,* 1957). Since the owner refused the virological examination for the presence of IBDV (as the parent flock was vaccinated against disease), and based on literature data, authors can only speculate on the possible causes and consequences. Regard to possible infection with the IBDV in 1-day-old chicks protected by maternal antibodies, it should be remembered that vaccination failure with inactivated vaccines is rare, but may occur (van den Berg *et al.,* 2000). **F**urthermore, the exposure to IBDV at the age younger than 1 week increases the susceptibility to e.g. MD (Giambrone *et al.,* 1976). On the other side, the appearance of hemorrhages in bursas in IBDV infected chickens is often, but not a consistent lesion (Eterradossi and Saif, 2013). Finally, but not less important, was the infection with *E. coli* (colibacillosis) confirmed in all the examined pullets aged 1 and 4 days. The haemorrhages in BF can be connected with some activity among red blood cells (RBCs) escaping through damaged blood vessels. Zeryehun *et al.,* (2012) assumed that such condition may be the consequence of disseminated intravascular coagulation (DIC). Except in the organisms with traumatic lesions or malignant diseases, in chicken DIC can be histologically confirmed in case of bacterial vasculitis (Power, 2000). Besides, here, the exact route of transfer of *E. coli* should be known, i.e., whether the bursa (if the examined chickens were actually infected by field IBDV!) could have previously been atrophied by a bacterial infection originating from the parent flock or the hatchery (Nakamura *et al.,* 1990), or the primary viral infection caused immunosuppression and then increased morbidity and mortality of chickens suffering from colibacillosis.

In all the examined poults aged 0-day (50; 65.79 % out of total 76 broiler turkey carcasses necropsied in 2014 / 2015), the most prominent macroscopic and histopathological lesions included degenerative changes in the pancreas (fibroplasia/fibrosis; necrosis); catarrhal (desquamative) duodenitis; myocardial degeneration followed by interstitial disseminated mononuclear cell hyperplasia; and atrophic changes and fibrosis in the bursa of Fabricius. Except omphalitis and/or yolk sac infections as the second prominent macroscopic findings confirmed in the pullets, histopathologic examination confirmed generalized arteriosclerosis in all 0-day (and 2-week) old poults (case 2).

Histopathologically, an incomplete development of the pancreas in the early postnatal period in chickens may be confused with pathological atrophy. However, in the present case the pathomorphological findings of partial atrophy was associated with fibroplasia/fibrosis, and in 1-day-old poultry the idiopathic (spontaneous) fibrosis of pancreas has not been confirmed so far. The pancreas, liver and the small intestine develop rapidly after hatching, emphasising the importance of these organs to the newly hatched chick (Katanbaf *et al*., 1988). The pancreas is one of the fastest growing organs in the first 10 days of chicken life (Phelps *et al.,* 1987). From the poor anamnestic data, it could be concluded that the poults with the pathomorphological diagnosis of size nonuniformity, pancreatic fibrosis and lymphoid organs atrophy and/or fibroplasia, originated from much younger parent flocks than the flocks with the offspring free of such lesions. These data suggest that the parent flock age is probably important for the development of the gastrointestinal tract and pancreas (Maiorka *et al.,* 2004). While after 1 week, the same authors could not see any morphophysiological differences, in the present case, pathomorphological lesions in the bursa, pancreas, intestine and the proventriculus were still present even in 2-week-old turkeys. Nitsan *et al.* (1991) wrote about a reserve of pancreatic enzymes in newly hatched chicks, which implied their production during the embryonic growth and the decline just after hatching. On the contrary, Sell *et al*. (1991) concluded that the total activity of pancreatic enzymes significantly increased after hatching. So, the continual findings of fibrotic changes in the pancreases of poults aged 0 day to 3 weeks examined in present investigation, could intimate the future problems with the pancreas exocrine function (MAS). The pancreatic atrophy and fibrosis can also be found in the second generation selenium-deficient chicks, but in the age 4–5 days and 14-16 days respectively, followed by a high mortality after 21 days of age (Cantor *et al.,* 1975).

Pathomorphological lesions resembled to lesions in RSS/MAS were associated with generalised arteriosclerosis in pullets in age 0 day and 2 weeks. These findings could be common in broiler turkeys toward the end of fattening period, especially as they are genetically hypertensive birds with extremely fragile blood vessel walls, living in conditions of intensive production of fattening. In the youngest pullets, (mostly intimal hypertrophic) arteriosclerotic changes, apart from the large and medium size arteries, were also observed in small arteries and arterioles (arteriolosclerosis) (Mitchell, 2015). Discussing genetic aspects of CVD in animals, Detweiller (1964) pointed out that, in contrast to studies of spontaneous arteriosclerosis in humans, a small number of animals is covered by the same research. It is therefore difficult to determine the main aetiological factor in each case of these chronic arterial diseases. This unsuspected histopathological finding became the main reason for starting  a systematic pathomorphological screening of several broiler turkey flocks during the whole period of fattening. Since broiler turkeys aged 8 – 19 weeks belong to the category of domestic poultry suffering from acute cardiovascular diseases (CVD) followed by a high mortality, the findings of such drastic degenerative changes in the arteries of the youngest pullets represent a real additional threat to the function of cardiovascular system over the whole period of fattening. Therefore, histopathologic findings of generalized arteriosclerosis in all the examined 0-day-old pullets represented a new challenge for the authors, not only in terms of identifying and quickly remedying the cause of pathological changes that could contribute to greater morbidity and mortality caused by acute cardiovascular disease (CVD) in broiler turkeys aged 8-19 weeks, but of opening of a new scientific approach to the research of the pathogenesis of CVD in broiler turkeys.

**Conclusion**

Based on very uncommon pathomorphological findings of haemorrhagic bursitis and RSS-type changes in 1 and 4-day old pullets, and RSS-type changes and generalized arteriosclerosis in 0-day-old poults – in addition of few diagnostic techniques that have been applied in accordance with the request of the owner - we concluded that it is necessary to continue the research the ethiopathogenesis of the natural occurence of hemorrhagic bursitis, the RSS-type changes and generalized arteriosclerosis in 0 and 1-day-old chicks and turkey poults. The main reason is the possibility of early age immunosupression which could be induced in these cases, but not less the possible genetic impact and the frequency of appearance the same changes in various hybrid lines through the systematic monitoring in future.

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**NEUOBIČAJENI PATOMORFOLOŠKI NALAZI U JEDNODNEVNE PERADI. PRIKAZ SLUČAJEVA**

Marina Tišljar1\*, Tajana Amšel Zelenika1, Vladimir Savić1, Borka Šimpraga1, Fani Krstulović1, Tihomir Zglavnik1

1Hrvatski veterinarski institut, Centar za peradarstvo, Zagreb, Hrvatska

\*dr. sc. Marina Tišljar, dr. vet. med., Hrvatski veterinarski institut, Centar za peradarstvo, Heinzelova 55, 10000 Zagreb, Hrvatska; tel.: +385 (01) 2440 214; fax: +385 (01) 2441 396; e-mail: tisljar@veinst.hr

**Sažetak**

**Prikaz slučajeva**

**Slučaj 1.**

Tijekom ljetnih mjeseci 2016. godine povećala se stopa smrtnosti u jatu mladih konzumnih nesilica. Patoanatomskom pretragom sveukupno 52 nesilice dobi jedan, četiri i 15 dana utvrđene su promjene karakteristične za sindrom zarazne kržljavosti (SZK) / sindrom malapsorpcije (SMA). U najmlađe je skupine (dob: jedan i četiri dana) patoanatomski nalaz ukazao na hemoragični burzitis (30; 57,69 %). U iste je skupine utvrđena upala pupčana otvora i žumanjčane vrećice. U 22 (42,31%) lešine nesilica dobi 15 dana prevladavale su promjene tipične za SZK. Patohistološkom je pretragom potvrđen patoanatomski nalaz. Bakterija *Escherichia coli* (*E. coli*) izdvojena je u svih pretraženih pilića. Prema osnovnim anamnestičkim podatcima vlasnika mlade su nesilice bile zaštićene od infekcije virusom zarazne bolesti Fabricijeve burze (ZBFB) maternalnim protutijelima te su, prema zahtjevu vlasnika, tijekom razudbe uzorkovani organi proslijeđeni isključivo na bakteriološku i patohistološku pretragu. U dostupnoj literaturi nismo naišli na opis spontane pojave hemoragičnog buzitisa i/ili patomorfoloških promjena tipičnih za SZK-i u jednodnevnih pilića.

**Slučaj 2.**

Od sveukupno 76 tovnih purana lešina različitih hibridnih linija, u 50 (65,79%) tovnih purana dobi 12-24 sata i 26 (34,21%) tovnih purana dobi jedan do dva tjedna patomorfološkom su pretragom utvrđene promjene tipične za SZK / SMA te upala pupčana otvora i žumanjčane vrećice u purana najmanje dobi. Promjene su pobudile sumnju na vertikalan prijenos SZK / SMA u tek izvaljenih purana. Patohistološkom su pretragom potvrđene promjene tipične za navedene sindrome, a u svih je purana utvrđena i generalizirana arterioskleroza. Uvidom u literaturne podatke dosad nisu pronađeni radovi o patomorfološkim promjenama koje bi ukazivale na spontanu pojavu SZK / SMA i generaliziranu arteriosklerozu u tek izvaljenih purana. Prema zahtjevu vlasnika od dopunskih je pretraga, osim patohistološke, učinjena i virološka pretraga metodama molekularne biologije.

**Zaključak**

U oba su slučaja opisane sponatno nastale patomorfološke promjene u jednodnevne peradi dosad neobjavljene u dostupnoj stručnoj i znanstvenoj literaturi. S obzirom na pojavu promjena tipičnih za SZK u jednodnevnih konzumnih nesilica i tek izvaljenih tovnih purana postavljena je sumnja na vertikalan prijenos bolesti. Potrebu za daljnjim istraživanjem potencijalno imunosupresivnih bolesti pobudio je i nalaz hemoragičnog burzitisa u jednodnevnih konzumnih nesilica zaštićenih maternalnim protutijelima od infekcije virusom ZBFB. Uzimajući u obzir neriješen problem ekonomskih gubitaka u proizvodnji tovnih purana muškoga spola oboljelih i uginulih od metaboličkih srčanožilnih bolesti akutnoga tijeka (puknuće aorte; sindrom nagle smrti i perirenalna krvarenja /osmi do 19. tjedan života/), do sada neutvrđen nalaz generalizirane arterioskleroze u purana najmanje dobi zahtijeva povećan opseg znanstveno-stručnog nadzora tijekom cjelokupnog razdoblja tova.

**Ključne riječi**: patomorfološki nalaz, tovni purani, jednodnevna konzumna nesilica, sindrom zarazne kržljavosti (SZK), sindrom malapsorpcije (SMA), hemoragični burzitis, zarazna bolest Fabricijeve burze (ZBFB), arterioskleroza