AN OUTBREAK OF HEMORRHAGIC DISEASE IN RABBITS

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Abstract
Rabbit viral hemorrhagic disease, known as viral necrotic hepatitis or hemorrhagic pneumonia, is a contagious disease with peracute or acute outcome and characterized by hyperthermia, prostration, hemorrhagic foamy discharge and mortality in 50-90% of cases. The main lesions are consisting in hemorrhagic diathesis, especially in lungs, liver and spleen. There are no evolution particularities regarding the age, breed, gender or species (domesticated or wild). The aim of this paper was consisting in describing the symptomatology and the pathology of hemorrhagic disease outbreak in rabbits. To the Faculty of Veterinary Medicine of Iasi were submitted for a physical examination 5 rabbits, 4 female and one male, crossbreed between Lionhead and Californian breed, 4 to 9 months old, kept in a private household, and. During the physical examination, the main symptomatology seen was consisting in sign of seizures, agitations, respiratory illness and nasal bloody and foamy discharge. Due to their bad conditions, the rabbits died shortly after the physical exam. The gross pathology was characterized by hemorrhagic diathesis, catarrhal-hemorrhagic rhinitis, laryngitis and tracheitis with the presence in lumen of a frothy and hemorrhagic liquid. The lesions seen in lungs were consisting in hemorrhagic pneumonia and edema, in liver a necrotic hepatitis, while in kidneys lesions with hemorrhagic pattern.
Keywords: *rabbits, hemorrhagic disease, lesions*

Introduction

The most serious infectious diseases of the rabbits are viral diseases, especially the hemorrhagic disease and mixomatosis.

Rabbit hemorrhagic syndrome is an infectious-contagious disease characterized by hemorrhagic diathesis in internal organs, hepatic necrosis, intravascular coagulation and high mortality. The causative agent is a virus of the *Lagovirus* genus, *Caliciviridae* family, which multiplies in the cytoplasm of host cells, in particular hepatocytes.

Domestic and wild rabbits are affected, irrespective of breed, gender or age. No other animal species is receptive to natural or experimental infection.

The most important sources of infection are represented by sick rabbits, rabbits with subclinical forms, which eliminate large amounts of virus. The infection expands quickly through direct contact, and the contamination is done by digestive and conjunctival ways and sometimes by cutaneous way trough lesioned skin.

Lagovirus replicates in the small intestine, liver and spleen lymphocytes.

In the present study, clinical signs, macroscopic and microscopic lesions were analyzed in an outbreak of hemorrhagic disease in rabbits, from a particular farm.

Materials and methods

The investigations were carried out on a herd of 34 rabbits, including 10 adults and 24 youngs of the Californian, Belgian Giant, German Giant, Butterfly and Alaska breeds, from a private farm, who presented for diagnosis at the Faculty of Medicine Veterinary of Iasi.

Rabbits were vaccinated with a bivalent vaccine and the first symptoms occurred in the summer of 2018, two months after vaccination.

Both adults and youngs showed clinical signs and specific lesions reported in the literature as belonging to the hemorrhagic disease.

A careful clinical examination was carried out. Necropsy and samples were collected for histopathological examination, in order to highlight the main lessions.

The organs were fixed in 10% formalin, then embedded in paraffin and cut at 5 μm thickness and stained with Trichrome Masson (Haematoxilin, Eosin and Blue methyl (HEA)), magnifying glass and photon microscope Leica ICC 50 HD, lenses x10, x20, x40.

Results and discussions

After the clinical examination of the 34 rabbits, the following clinical aspects were identified: pronounced state of prostration, lack of appetite, lateral torsion of the head, seizures, sometimes nasal bleeding (Fig. 1) and death.

These symptoms have been present in the herd for two weeks, during which all rabbits have died. Due to the rapid evolution and serious symptoms we can conclude that, there were superacute and acute forms of disease.

Following the necropsy examination of the cadaver, lesions were observed in the liver (Fig. 2), lung and spleen. The liver showed a faded appearance and some diffuse necrosis.

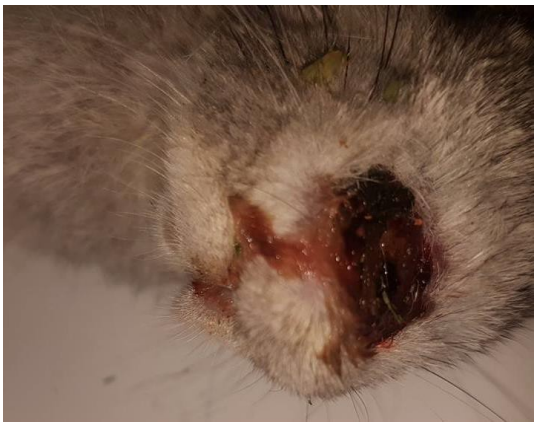

Fig. 1 Clinical aspect (Nasal bleeding)

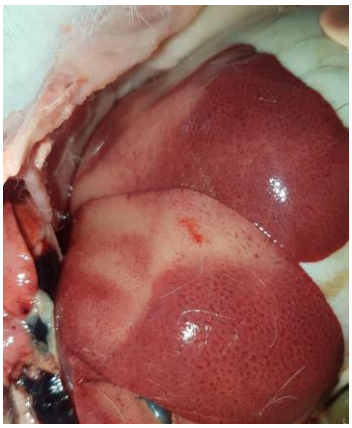

Fig. 2 Clinical aspect (Faded liver)

Pulmonary congestion and hemorrhage were observed in the lungs (Fig. 3). Other lesions observed in the respiratory tract were represented by diffuse hemorrhages in the larynx and trachea and hemorrhagic exudate (Fig. 4).

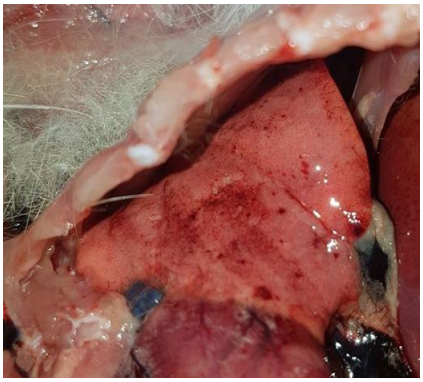

Fig. 3 Congestion and pulmonary hemorrhage

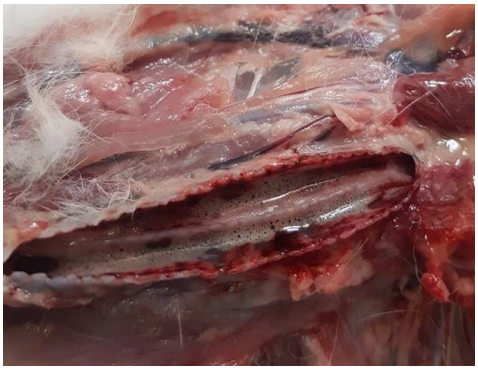

Fig. 4 Haemorrhagic lesions in the trachea

In the urinary tract, there were also highlighted hemorrhagic lesions: bleeding in the urinary bladder wall and hemorrhagic lesions on the kidney surface.

After the animals death, biological samples from all organs with lesions (liver, kidney, trachea, lung, heart) were collected in order to perform the microscopic examination.

The histopathological examination of the main organs identified with lesions on the necropsy exam revealed the following lesions: liver with obvious circulatory disorders such as congestion, hemorrhage, micro- and macrotrombosis.

The hepatocytes presented necrobiosis phenomena (cariorexis, cariolysis), jaundice, hemosirerophage and oxyphyl inclusions in the cytoplasm (Fig. 5, Fig. 6); the kidneys presented interstitial hemorrhages, proteinuria, hyaline cylinders in the lumen of contour tubes and glomerular hyalinizations, serum-hemorrhagic glomerulonephritis, perivascular and interstitial edema (Fig. 7, Fig. 8). In the trachea, venous congestion was observed (Fig. 9).

The lungs showed hyperemia, hemorrhage and edema around the bronchioles, bronchi, alveoli, as well as micro- and macrotrombosis (Fig. 10, Fig. 11). The cord exhibited hemorrhages located in myocardial thickness and intramuscular hyalinosis (Fig. 12).

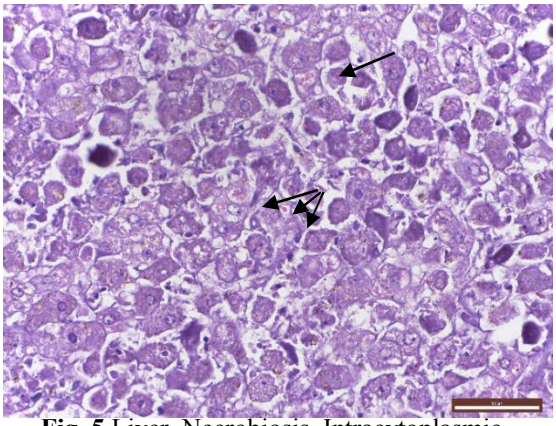

Fig. 5 Liver. Necrobiosis. Intracytoplasmic inclusions, hemosiderin, edema. Trichrome Masson stain

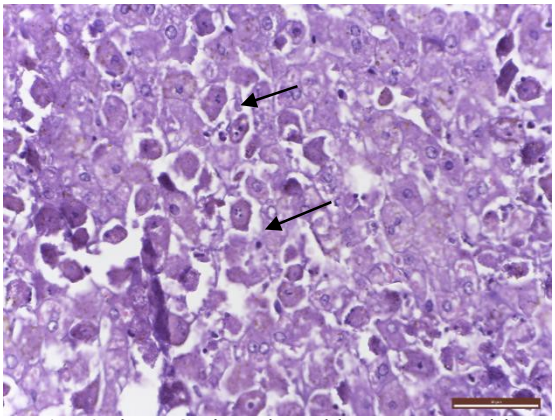

Fig. 6 Liver. Cariorexis and hepatocytes with cariolysis phenomena. Trichrome Masson stain

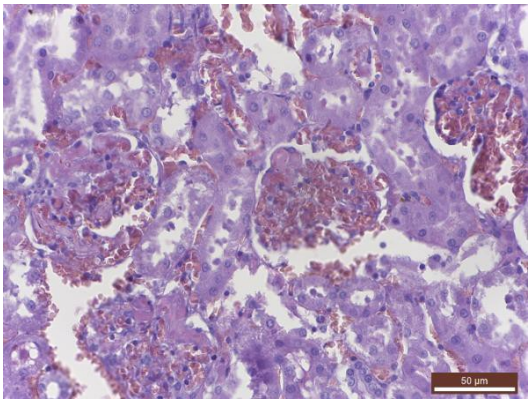

Fig. 7 Kidney. Congestion and glomerular haemorrhage, hyalinization of glomerular capillaries. Trichrome Masson stain

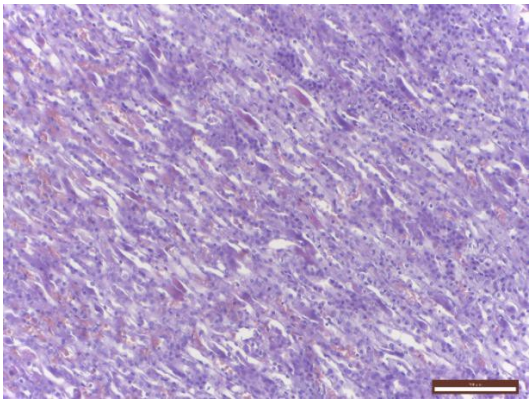

Fig. 8 Kidney. Atrophy, congestion and hyaline cylinders. Trichrome Masson stain

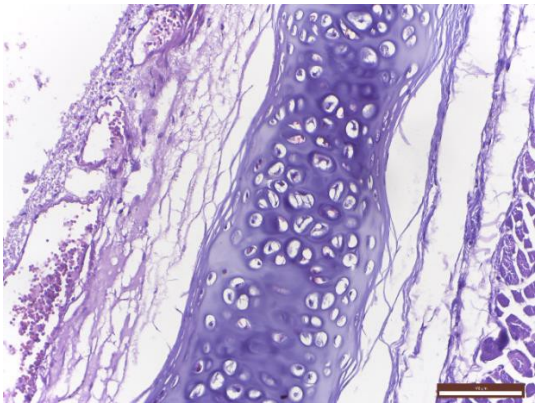

Fig. 9 Trachea. Venous congestion. Trichrome Masson stain

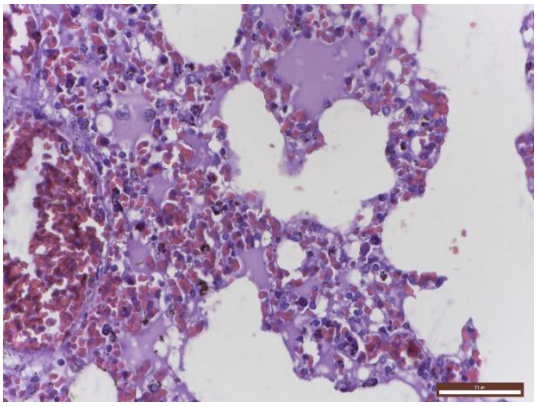

Fig. 10 Lung. Congestion, microtrombosis, pulmonary edema. Trichrome Masson stain

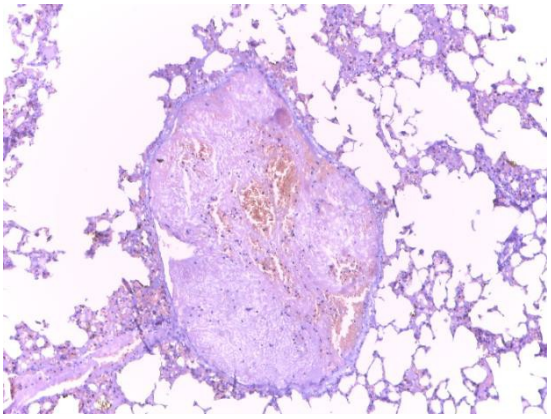

Fig. 11 Lung. Macrotromb. Trichrome Masson stain

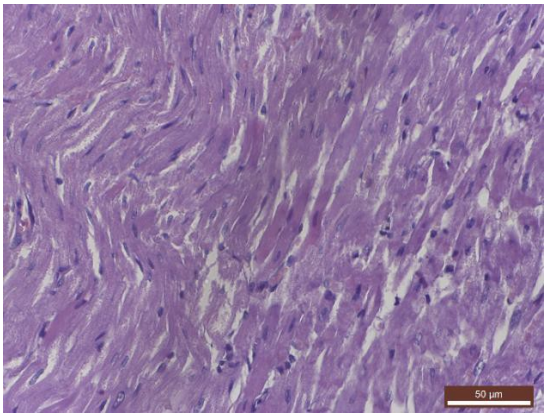

Fig. 12 Heart. Hyalinization of myocardiocitis. Trichrome Masson stain

Conclusions

The main clinical aspects identified in the hemorrhagic disease outbreak have been clustered in superacute and acute forms.

The most pronounced hemorrhagic lesions were observed in the liver, respiratory system (lung, trachea) and heart.

Histopathological lesions dominant in the rabbits hemorrhagic disease were represented by circulatory disorders (congestion, hemorrhage, edema, micro- and macrotrombosis), cellular and nuclear necrosis phenomena, hepatocellular jaundice and intracytoplasmic inclusions.

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