Morphoclinical Aspects In Experimental Haemorragical Disease Virus Of Rabbits

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Abstract. Rabbit haemoragic viral disease is a virotic infections disease highly contagious, with an acute evolution, characterised by fever, haemoragic sindrome, necrotic hepatitisis and high morbidity and mortality.

The disease may ocur as a natural infection or an experimental one. The aim of this study is to prevent some morphoclinic aspects in an experimental infection with haemoragic virus on rabbits.

Key words: rabbits, haemoragical, liver, disease.

MATERIALS AND METHODS

The experiment was concluded on 15 rabbits 6 months age, not vaccinated against haemoragic disease, infected via intranasal way.

After exitus, the rabbits were necropsied and the falling organs were prelevated: heart, kidneys, lungs and liver. The samples were histophatologic prepared: phormaldehide 10% fixation, paraphine inclusion, microtome sectioned (6 µm) and stained by HEA method.

RESULTS AND DISCUSSIONS

Four rabbits died after 42-45 hours post infection. The other eleven died after 45-48 hours post infection.

Only three rabbits showed clinical signs before exitus, the other twelve didn’t show any signs, due to the supraacute evolution of disease.(1)

The clinical signs observed were: nasal haemoragic dispneea, nervous signs, acompanied in the agonic phase of the disease by restless and strident screems.(Fig.1)

All rabbits were necropsiated by mamallic techniques.

Macroscopic examination reveals septicemic haemoragical lesions. Congesties and haemoragies on lungs, haemoragic infiltration at traheal and bronchial mucous in traheic and bronhic luminen was observed an abundant haemoragic exudation.(Fig. 2,3,4) (3, 6)

Liver presented necrotic hepatitis, the organ had increased volume and weight with a yellow colour and a friabil condition.(Fig5)

The hepatic lesions and disseminated intravasculary coagulation are the most important in the disease evolution, the haemoragical virus having affinity for the hepatic cells, the virus multiplies itself in their cytoplasm leading to their necrosis.

The hepatic necrosis are the key element in disease phatogenesis, the necrosis activate the coagulation of the blood factors (one of the most important in the liver is the synthesis of mucous proteins witch interfere in the coagulation mechanism.)
By virus multiplication in hepatic cells emerge some disturbances in the coagulation factor synthetisis and consecutive generalized haemoragic lesions.

The initial hepatic necrosis appeared due to viral multiplication inside the hepatic cells, fact witch switch out a generalized microtrombosis, this one will accelerate the hepatic necrosis, consequently to the ischemia produced by microtrombs.2, 3)

The kidneys are strongly congested, with increased volume and weight, with friabile consistency, by sectioning the kidney a red-cherry coloured blood is draining.(Fig.6)

Histopathologic examination reveals diffuse necrosis of liver cells, the presence of hemosiderin in Kupffer cells, hemorrhagic infiltration in lungs and limfohistiocitar infiltration on kidney corticala.(4, 5)
Picture 1. Clinical aspects. Nasal haemorrhagia

Picture 2. Tracheal haemoragical exudation

Picture 3. Lung haemorrhagia and congestion.

Picture 4. Lung haemorrhagia and congestion.

Picture 5. Necrotical hepatitis

Picture 6. Renal congestion
CONCLUSIONS

The clinical signs observed on infected rabbits were: nasal haemoragia, dispneea nervos signs and in the organic phase excitement signs and shouts.

The macroscopic examination reveals septicemic type haemoragic lesions.

The lungs presented congestia and hemorrhagia, at the mucous level.

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REFERENCES


