# MORPHOCLINICAL ASPECTS IN EXPERIMENTAL HAEMORRAGICAL DISEASE VIRUS OF RABBITS

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**Summary**

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 fallowing 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, acompaniated 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 and hemorrhagic lesions. Congesties and hemorrhages on lungs, hemorrhagic infiltration at traheal and bronchial mucous in trachea and bronhic lumenum 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 intravascular 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 is 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).

Histophatologic examination reveals diffuse necrosis of liver cells, the presence of hemosiderin in Kupffer cells, hemoragic infiltration in lungs and limfohistiocitar infiltration on kidney cortical(4, 5).

## Conclusions

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

The macroscopic examination reveals septicemic type hemorrhagic lesions.

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

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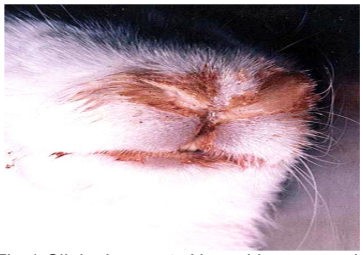

Fig 1.Clinical aspects.Nassal haemorragia

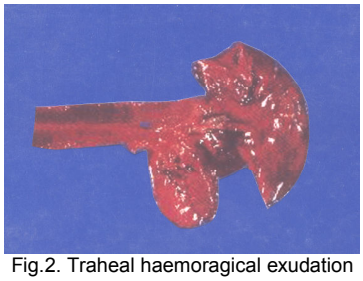

Fig.2. Traheal haemoragical exudation

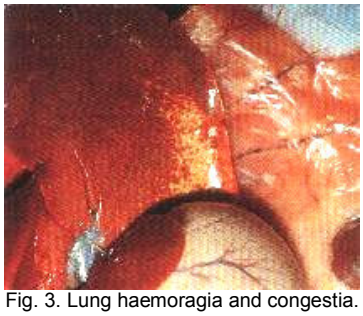

Fig. 3. Lung haemoragia and congestia.

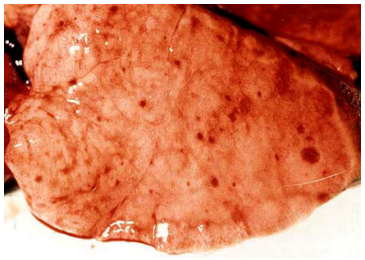

Fig.4. Lung haemoragia and congestia

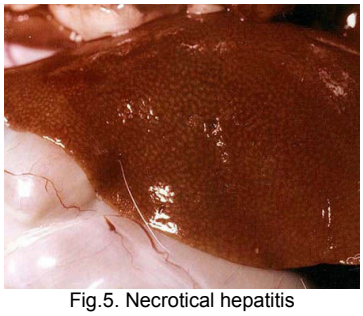

Fig.5. Necrotical hepatitis

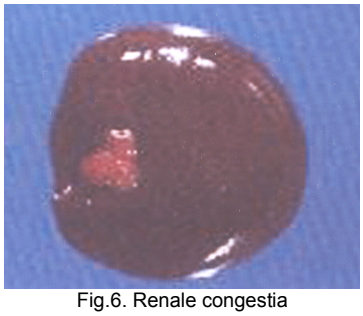

Fig.6. Renale congestia

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