

SUMMARY OF THESIS*

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INTERNALIZATION OF IMMUNOGLOBULINS BY ENDOTHELIAL CELLS IN THE LIVER, LUNG AND KIDNEY IN HAMSTER WITH VISCERAL LEISHMANIASIS

During visceral leishmaniasis several organs are affected but the pathogenic mechanisms are not fully known. In this study we evaluated the participation of immunoglobulins in the liver, lung and kidney lesion in visceral leishmaniasis. In hamsters injected intraperitoneally with 2×10^7 amastigotes of *Leishmania (Leishmania) chagasi* we observed progressive increase of parasite burden in the spleen and liver and the anti-*Leishmania* antibody titer in serum during infection. IgG deposits were detected by immunoenzymatic test in the liver outlining the sinusoids and in the lung in alveolar walls. No significant C3 deposits were observed either in the liver or in the lung when compared with non infected controls. Since both localization of IgG deposits and absence of C3 deposits were not compatible with immune complex deposition we examined the kidney and we observed IgG deposits around the glomerular capillary walls and in the tubuli and weak C3 deposits. IgG deposits in all organs were more intense at 30 and 45 days post-infection (PI). The histopathological analysis in the liver, lung and kidney showed progressive alterations but with change in the composition of inflammatory infiltrate suggesting participation of different pathogenic mechanisms in different phases of infection. *Leishmania*

antigen was detected in organs within macrophages and as extracellular particulate material. Since observation under optic microscopy was not compatible with deposition of immune complex, we studied the internalization of immunoglobulins by endothelial cells, then we analyzed qualitative and quantitatively the presence of immunoglobulins, ultrastructurally, in these cells. In the liver Disse's space enlargement and greater amount of immunoglobulins in endothelial cells, in Disse's space and in sinusoid were observed at 30 days PI when compared with non infected controls. In the lung we observed significantly greater amount of immunoglobulins at 30 days PI in the endothelial cells when compared with that at 60 days PI. In the kidney we observed significantly greater amount of immunoglobulins at 30 days PI in endothelial cells in glomeruli and in the tubular region and in tubular epithelial cells at 30 days PI when compared with non infected controls. Our results show deposition and internalization of immunoglobulins in the endothelial cells in visceral leishmaniasis in hamsters. This phenomenon was prominent around 30 days of infection and this may be an alternative mechanism of lesion in visceral leishmaniasis.

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