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PATHOLOGICAL STUDIES OF RATS INFECTED WITH
HEMORRHAGIC FEVER WITH RENAL SYNDROME (HFRS) VIRUS

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To elucidate the pathogenesis of renal and nervous lesions in the zoonotic disease, hemorrhagic fever with renal syndrome (HFRS), histopathological, immunohistochemical, and ultrastructural examinations were performed mainly on the brain (chapter 1) and kidney (chapter 2) tissues of SPF rats infected with strain SR-11 (SR) of HFRS virus. New born SPF rats were inoculated intraperitoneally with the SR virus, and pathological observations were carried out from 3 to 20 days after infection.

The results are summarized as follows.

Chapter 1.

The infected rats exhibited neurological symptoms such as slight tremor and ataxia, from 11 days postinoculation (PI). Histologically, focal degeneration and necrosis of nerve cells with eosinophilic cytoplasmic inclusion bodies (CIB) were observed in the cerebral cortex and hippocampus on and after 11 days PI. CIB were positive for the viral antigen. Ultrastructurally, CIB were granular, filamentous, and granulofilamentous in structure and were located near the well-developed Golgi apparatus with increased ribosomes. CIB were rarely observed in oligodendrocytes and macrophages. From these findings, it was considered that HFRS virus is highly neurotropic, and synthesis and accumulation of the viral protein (CIB) cause the nerve cell damage.

Chapter 2.

The infected rats exhibited proteinuria on and after 16 days PI. Histologically, the epithelial cells of the proximal renal tubules showed slight vacuolar and granular degeneration with the same CIB as described in chapter 1 from 15 days PI. Ultrastructurally, decrease of endocytic vesicles, invaginations of the apical cytoplasmic membrane, and an irregular arrangement of the brush border were observed in the swollen epithelial cells of the proximal renal tubules. On immunohistochemical examination, CIB were positive for anti-SR nucleocapsid antibody, but negative for anti-SR envelope protein antibody. These findings reveal that the proteinuria resulted from tubular damage due to the viral infection, and CIB consisted of the viral nucleocapsid.

From these results, it is concluded that this experimental system is suitable for the study of the pathogenesis of the nervous and renal lesions in HFRS.