PERSISTENCE OF COXSACKIE A13 VIRUS IN BALB/C MICE WITH T-CELL DEFICIT

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Received December 20, 1985

The mice with T cell deficit resulting from natural thymus atrophy (athymic mice) or from the administration of antithymocyte serum and infected with Coxsackie B3 virus appeared to have a decreased intensity of myocardial inflammatory changes. The available data on the persistence of the virus in these models are controversial (*1—4*).

We studied the effect of T cell deficit on the course of infection with Coxsackie A13 virus (prototype strain Flores) in Balb/c mice. ALS was raised in rabbits against Balb/c mice spleen lymphocytes (cytotoxicity titre 1 : 800). It was administered in 3 i.p. doses at 0.2 ml. Athymic Balb/c mice were supplied by E. V. Gruntenko (Institute of Cytology and Genetics, Siberian Branch of the USSR Academy of Medical Sciences). Intact animals of the same line as well as mice pretreated with normal rabbit serum (NRS) were used as controls. Coxsackie A13 virus grown in human embryo fibroblasts was administered i.p. in a single dose of 0.2 ml (titre of 4.5 $\log_{10}$ TCD_{50}/ml). Mice were sacrificed on days 7 and 21 p.i. and 10% suspensions were prepared from their internal organs. The suspensions were inoculated into NEr 2 cells. The reisolated strains were identified in neutralization test using standard antiserum (from the Institute of Poliomyelitis and Viral Encephalitides, U.S.S.R. Academy of Medical Sciences, Moscow).

In all 20 infected mice pretreated with ALS, the virus was detected in the heart, liver, kidneys, spleen and lymph nodes at both intervals. The infectious titre was 2.56—3.23 on day 7 and 1.9 $\log_{10}$ TCD_{50}/ml on day 21. In mice, which were given virus alone or virus after NRS treatment, no virus was isolated by day 7 p.i. In infected athymic mice the virus was detected on day 7 in the heart, liver, spleen, lymph nodes and blood clot. The virus titre was in no case higher than 2.9 $\log_{10}$ TCD_{50}/ml. On day 21 the virus was detected only in the heart, liver and spleen at a titre of 1.0 $\log_{10}$ TCD_{50}/ml.

Pathomorphological examination of the myocardium of athymic or ALS-treated mice has revealed a marked decrease of intensity of necrotic and inflammatory changes as compared to normal mice. Our findings are consistent with the data (*3*) on persistence of Coxsackie B3 virus for 28 days in the myocardium of NFR nu/nu mice. Our results as well as the available literature indicate that T lymphocytes play an important role in the control of virus infection and pathogenesis of virus induced lesions.

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