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Choline acetyltransferase (ChAT) activity in porcine brains after Encephalomyocarditis (EMCV) virus infection

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Clinically, EMCV causes a sudden death syndrome in piglets due to acute myocarditis. EMCV might be considered a zoonotic agent, as antibodies to the virus have been demonstrated also in human beings, however, there are only few reports of potentially clinical effects of the virus to humans. Interestingly, in rats *in vitro* studies have demonstrated an age dependent degeneration in Hippocampus after EMCV infection. This study suggested the development of neurodegenerative processes due to the presence of EMCV or infection. Acetylcholine plays a role in neurodegenerative processes and memory in the central nervous system (CNS). Choline acetyltransferase (ChAT), a marker for cholinergic neurons, is widely distributed in mammalian brain and humans. Reduction of cholinergic activity has been demonstrated in animal experimental epilepsy or in neurodegenerative human disorders as in Alzheimer's disease. In this study we searched the alterations of ChAT activity in different brain regions after EMCV infection of piglets. Piglets with an age of 8 weeks were inoculated intranasal and peroral with the EMC Virus (8 ml, EMCV strain B279/95, 10^7 TCID 50/ml) or medium (control animals, CO). Clinical exams were carried out twice a day to verify the infection state. On day 6 post infection the piglets were sacrificed the peripheral organs and the brain was immediately removed. Brain regions i.e. frontal cortex, parietal cortex, temporal cortex, hippocampus, caudate nucleus, putamen, globus pallidus, thalamus, hypothalamus, substantia nigra, cerebellum, bulbus olfactorius, midbrain, pons and medulla were prepared on ice and frozen at –80°C until analysis. By using radioenzymatic method (3, 4) we analysed the activities of ChAT in porcine brain regions after EMCV infection (N=4) and in corresponding CO (N=4). For pathohistological investigation, samples from heart and brain of EMCV infected piglets and corresponding controls were fixed in 7% buffered formalin. Six days after EMCV infection marked histopathological changes were revealed in heart tissue. All EMCV-infected piglets showed a diffuse myocarditis simplex with perivascular cell infiltration and dystrophic calcification. In CO piglets, highest activity of ChAT was found in putamen (13.8 nmol/mg wet weight tissue/h) followed by caudate nucleus (11.1 nmol/mg wet weight tissue/h) and midbrain (5.6 nmol/mg wet weight tissue/h) and the lowest activity of ChAT was measured in cerebellum (0.2 nmol/mg wet weight tissue/h). Six days after EMCV infection we found significantly lowered ChAT activity in midbrain (54% of CO; p=0.007), medulla (56% of CO; p=0.03) and bulbus olfactorius (84% of CO; p=0.01). In other brain regions a moderate reduction (10-15% of CO) was revealed, whereas in the thalamus ChAT activity was lightly increased (111% of CO). Present study provides evidence that EMCV infection significantly affected cholinergic activity in the CNS. The reduction of the neuronal marker ChAT gives suggestion for degeneration of cholinergic neurons. In summary we can say that EMCV infection in piglets is accompanied by significant pathological changes involving also the brain. Lowering of cholinergic activities in the brain can be in part of importance for development of depression and occurrence of sudden death syndrome. With respect to opportunistic infections in HIV-1 infected patients this also might form the pathology of the AIDS-dementia syndrome.

Biography

B Kepplinger was Medical Director of the neuro-rehabilitation Wall Amstetten and the Neuro Rehabilitation and Pain Wall Amstetten and Head of acute Logie and Stroke Unit. In the past 10 years, he worked as medical director of the State Hospital Wall Amstetten.

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