COXSACKIEVIRUS GROUP B TYPE 4 INDUCED MYOCARDIAL INFARCTIONS ?*

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Coxsackieviruses Group B (CBV) have been implicated in human cardiovascular diseases, predominantly viral myocarditis *(1)*. Seroepidemiological evidence has implicated CBV in myocardial infarction (MI) *(2)*. Coxsackievirus Group B may play either a casual or initiating role in some cases of MI. In a recent review Reyes and Lerner discussed the effects of CBV in experimentally induced murine myocarditis *(3)*. The gross and histopathologies induced by CBV during acute and convalescent post-infection (p.i.) periods were evaluated. The degree of response varied among strains of mice and was dependent on strain haplotype. Severe acute infections (8 days p.i.) generated extensive necrosis, mononuclear cellular infiltration, fibrosis, and mineralization. Lesions were always greatest in male animals. Chronic changes included fibrosis, mononuclear infiltrate, calcium deposition, artrial thrombi and aneurysms in both ventricles and septum. Lesions were more severe on animals forced to remain active during convalescence. Fifty per cent of these animals died within fifteen months of exposure. The experimental interval of observation in this study was eight days p.i. until fifteen months. Whether cardiopathologies reminiscent of MI occur early in CBV infection remains undetermined.

During recent experiments aimed at elucidating the variation among Coxsackievirus Group B type 4 (CB4) variants we isolated several viruses capable of generating rapid lethal infections in adult male Balb/c mice *(4)*. All animals infected with 1×10^6 virions died within 3 days. Animals were carefully monitored and immediately upon death, hearts were removed, hemisected longitudinally and one half preserved for histopathologic analysis and the other saved for viral isolation. Large amounts of virus were recovered from the hearts of all animals (10^4/gram). Random sections through the hearts revealed large areas of coagulative necrosis. These areas were histologically reminiscent of tissue recovered after acute fatal MI in humans. The behavior of the animal prior to death suggested neurologic involvement was minimal, if existent. The rapid and fulminant nature of this infection suggest cardiac involvement played a significant role in their demise. We are currently working to determine the extent of these lesions and plan extensive experimentation to determine why only specific isolates of CB4 produce this lesion.

Although current studies are not completed, dissemination of information regarding this viral-induced lesion to our colleagues appeared warranted. If CB4 viral-induced myocardial infarctions do occur in humans, it will be difficult to document the virus as the causative agent. CBV clears the body rapidly, possibly before the damage they have done is clinically apparent. Documentation of CBV concominant with MI will require early blood cultures or cultures from the infarcted area in rapidly fatal cases. Furthermore, as children are most often the victim of generalized CBV infection, infarcted, or atypical areas of myocardium from children dying of CBV infections should be cultured for virus and these specific viral isolates collected and saved for future evaluation in animal models. Lastly, the information provided by Reyes and Lerner and this study requires a careful reflection when caring for children recovering from CBV infections.

References

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