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INFLUENCE OF SEQUENTIAL REINFECTIONS WITH DIFFERENT CLONES OF THE COLOMBIAN STRAIN OF TRYPANOSOMA CRUZI ON MURINE CHRONIC MYOCARDITIS.

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Introduction: reinfections with Trypanosoma cruzi in patients living in endemic areas is considered as a factor of aggravation of Chagas disease myocardopathy. Experimentally, there are an increase of myocarditis and myositis in mice successively inoculated with strains of different biotypes. Predominance of the same type of strain, in one endemic area, representing a principal clone, suggests the possibility of multiple infections with the same strain. Objective: to investigate the influence of reinfections with clones with different degrees of virulence, of the Colombian strain, on chronic myocarditis in mice. Material and methods: Swiss mice infected with clones of the Colombian strain Biotype III, Triatul: I Col-C1 (high virulence); Col-C5 (medium virulence); Col-C8 (low virulence). Infected mice were sacrificed for homopathology in the acute phase (0, 20, 30 days p.i) and in the chronic phase of infection (150 days), with exception: of the infected with Col-C1, that did not survive until the chronic phase. The group submitted to triple infection was evaluated 115,130, 175 days after first infection. Enzootic for all groups 5 x 10^5 blood forms of T. cruzi intraperitoneally. Sections of the heart and skeletal muscle fixed in 10% Formalin, paraffin embedded and 5 µm sections stained with Hematoxylin and Eosin and Pico-Sirius method. Humoral response was investigated by indirect immunofluorescence and by Elisa tests and the delayed hypersensitivity (DTH) by cutaneous test. Results: At the acute phase, (single infection) inflammatory lesions of the heart varied from mild to moderate, with necrosis of cardiac myocytes. Mice with triple infection presented lesions that varied, from moderate to intense in the heart and skeletal muscle, on the 25th and 30th days after the triple infection, more intense than the animals submitted to a single infection. Mice with triple infection evaluated on the 175th day had chronic myocarditis in all cases, with lesions that varied from mild to moderate. Serologic titers varied from 1/10 to 1/640 in the single infection groups and from 1/10 to 1/2560 to triple infection. Immunoglobulins levels IgM and IgG were more elevated on animals submitted to triple infection. The evaluation of DTH was positive, with a higher intensity of the lesions in the period of 48 hours in the animals with triple infection and 24 hours in the single infection. Our results confirmed that the mice submitted to multiple infections presented an increase of inflammatory lesions, higher levels of immunoglobulins and serologic titers, when compared to those with single infection. Conclusion: Reinfections with T. cruzi clones contribute to an aggravation of the evolution of Chagas myocardopathy in the murine model of Chagas disease, even when reinfections were performed with clones of the same strain, with different degrees of virulence.