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Main Cardiac Histopathologic Alterations in the Acute Phase of *Trypanosoma cruzi* Infection in a Murine Model

Symptoms in the acute phase of Chagas disease are usually mild and nonspecific. However, after several years, severe complications such as dilated heart failure and even death may arise in the chronic phase. Due to the lack of specific symptoms in the acute phase, the aim of this work was to describe and analyze the cardiac histopathology during this phase in a CD1 mouse model by assessing parasitism, fibrotic damage, and the presence and composition of a cellular infiltrate, to determine its involvement in the pathogenesis of lesions in the cardiac tissue. Our results indicate that the acute phase lasts about 62 days post-infection (dpi). A significant increase in parasitemia was observed since 15 dpi, reaching a maximum at 33 dpi (4.1×10^6). The presence of amastigote nests was observed at 15–62 dpi, with a maximum count of 27 nests at 35 dpi. An infiltrate consisting primarily of macrophages and neutrophils was found in the cardiac tissue within the first 30 days, but the abundance of lymphocytes showed an ≥ 8 -fold increase at 40–62 dpi. Unifocal interstitial fibrosis was identified after 9 dpi, which subsequently showed an ≥ 16 -fold increase at 40–60 dpi, along with a 50% mortality rate in the model under study. The increased area of fibrotic lesions revealed progression in the extent of fibrosis, mainly at 50–62 dpi. The presence of perivasculitis and thrombus circulation disorders was seen in the last days (62 dpi); finally, cases of myocytolysis were observed at 50 and 62 dpi. These histopathological alterations, combined with collagen deposition, seem to lead to the development of interstitial fibrosis and damage to the cardiac tissue during the acute phase of infection. This study provides a more complete understanding of the patterns of histopathological abnormalities involved in the acute phase, which could help the development of new therapies to aid preclinical testing of drugs for application in Chagas disease.

Keywords: acute Chagas disease, cardiac histopathology, pathogenicity, mouse model.



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Biography

Dr. Paz María Silvia Salazar Schettino is a full professor emeritus in the Department of Microbiology and Parasitology at UNAM, a member of the National System of Researchers (SNI), and a titular member of the Mexican Academy of Sciences, with 55 years of scientific and teaching career. She has led research on Chagas disease, publishing over 123 articles, two books, and 31 book chapters. She has trained numerous graduate students, coordinated national and international projects, and received multiple honors, including the Sor Juana Inés de la Cruz Medal (2013).