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Air pollution on myocardial remodeling in acute phase of Chagas disease in experimental model.

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Background: There are more than 8 million patients with Chagas’ disease around the world. This disease is characterized by intense myocardial fibrosis triggered by a complex cascade of inflammation, oxidative stress and apoptosis. The air pollution is a health problem in urban centers and stimulates the same pathways. We aimed to assess the role of air pollution upon myocardial fibrosis in acute phase of Chagas’ cardiomyopathy model. Methods: 100 females Sirius Hamsters were divided into 4 groups: Control (Ct), Control + Pollution (CtP), Chagas (Ch) and Chagas + Pollution (ChP) and analyzed until sixty days of infection. The animals were infected with 10⁵ Tripanossoma cruzi Y strain. Animals were daily exposed to pollution by inhalation of particulate matter produced by burning diesel fuel. The animals were euthanized after 60 days. Morphometric analyses of the interstitial (ICVF) was performed. The evaluation of the inflammation, oxidative stress and apoptosis were studied by gene expression analysis using real-time RT-PCR, ELISA and TUNEL. Results: In the analysis of ICVF, we observed a higher deposition in the LV of the chagasic groups compared to control (p=0.04). Also pollution alone increased myocardial fibrosis in the control exposed to pollution compared to the control group. We observed that the infection did not amplify the deposition of interstitial collagen in the infected groups. Oxidative stress analysis Nox1, MnSOD and iNOS showed higher expression in the Ch and ChP compared to controls, the pollution did not modulate an expression of these genes in infected animals. In the analysis of apoptosis, we observed that the infected groups showed higher expression of the Bcl-2 and Caspase 3 genes in comparison to the controls. The ChP group showed even greater expression than the Ch group suggesting amplification of apoptosis modulated by pollution. In the protein analysis of the inflammatory cytokines on tissue IL-10 and INF-? we observed increasing of these cytokines in the infected groups. TNF-a also increase, but with no statistical significance. Chagasic group exposed to pollution presents 3 times more apoptotic cells than the chagasic group, demonstrating again a possible modulation of apoptosis by pollution. Conclusions: We concluded that air pollution amplified apoptosis in a Chagas heart disease model acute phase. However, it was not enough to increase myocardial fibrosis, probably because the strong pathway activation by T. cruzi infection.