Chagas disease and its complications

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Albeit generally known to be broad in Latin America, due to recent trends in migration, there are millions of people from Chagas disease endemic countries now living in North America, Europe, Australia and Japan, including thousands of people with Trypanosoma cruzi infection. Most infected individuals are not aware of their status. Congenital, transfusion- and/or transplant-associated transmission has been documented in the United States, Spain, Canada and Switzerland; most instances likely remain undetected. High priorities include the implementation of appropriate screening, evaluation and clinical management, and better assessment of the true burden associated with this disease. Therefore, Chagas disease should be considered in the differential diagnosis of cardiac problems such as acute myocarditis [1, 2].

Apart from its well-defined cardiac manifestations, Chagas disease is also known to trigger anti-neuronal autoimmunity resulting in neurodegenerative disease [3, 4]. Reactive oxygen species as well as inflammatory cytokines and a lot of other molecular mediators are reported to be involved in the pathophysiological processes caused by Chagas disease (or T.cruzi infection)[5]. However, the details of molecular interactions causing the systemic manifestations in Chagas disease need still to be elucidated. The current number of published randomized controlled trials is limited and more experimental studies have to be executed in order to clarify the underlying mechanisms of this clinical issue.

In this issue of the “Journal of Experimental and Integrative Medicine”, Chumbinho et al once again brought up the complications associated with Chagas disease in their well-defined experimental work concerning the cardiorenal axis of T.cruzi infected mice [6]. This study provides new evidence for the importance of the renin-angiotensin-aldosteron system during Chagas disease; briefly, an opposite effect of angiotensin and aldosteron was detected where angiotensin supports the heart and minimizes kidney injury but aldosteron aggravates heart failure. In an earlier report of Botoni et al in a small trial with randomly assigned 42 chronic Chagas cardiomyopathy patients to receive either the non-selectif beta-blocker carvedilol or placebo in addition to the angiotensin converting enzyme (ACE) inhibitor enalapril and spironolactone (an aldosterone antagonist) treatment, the regimen of carvedilol plus ACE inhibitor and aldosterone antagonist resulted with improvements in the heart failure score and quality of life [7].

Taken together, a clinical study reporting benefit of a ‘beta-blocker + ACE inhibitor + aldosterone antagonist’ regimen [7], but the -at least in part-contradictory outcome with increased mortality when the angiotensin II receptor type 1 (AT1R) was blocked [6] underlines the need for more research on this area to find out the ideal therapeutic approach in patients with T.cruzi-induced Chagas manifestations.

References


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