

LETTER TO THE EDITOR

Bilateral sympathectomy in a dilated cardiomyopathy model

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Cardiovascular diseases are still the main cause of death in the world. Among those, the leading indication to cardiac transplantation is dilated cardiomyopathy (DCM). This disease is caused by a variety of etiologies and culminate in ventricular remodeling, characterized by dilation of the ventricular chamber and systolic dysfunction, leading to congestive heart failure¹.

Ventricular remodeling involves activation of the sympathetic and renin-angiotensin-aldosterone systems. As such, classic treatments include the use of beta-blockers, angiotensin-converting enzyme inhibitors (ACEi) and mineralocorticoid receptor antagonists². As those treatments are continuous and only delay the development of the disease, it is important to look for better therapeutic options. In this sense, experimental models demonstrated benefits of sympathetic blockade on preserving ventricular function and preventing left ventricle remodeling after acute myocardial infarction³.

As presented in the XXXVII COMU in 2018, we evaluated the repercussions of the surgical inhibition of the sympathetic nervous system through bilateral sympathectomy and the clinical treatment with ACE inhibitors on dilated cardiomyopathy induced by doxorubicin in rats.

For that, Wistar rats were randomly divided into 4 groups, being: Control, BS, ACEi and a Sham group

(negative control). The first 3 groups underwent induction of DCM by intraperitoneal injection of doxorubicin, while Sham group received intraperitoneal 0.9% saline solution weekly doses. At the fifteenth day after induction start, BS group rats were submitted to irreversible chemical sclerosis of the starlit ganglion through ethanol injection. From the same period, animals in the ACEi group received daily enalapril diluted in oral water until the end of the experimental protocol. Ten weeks after the beginning of DCM induction, four parameters were evaluated: left ventricular (LV) function with the analysis of pressure-volume loops, quantification of fibrosis degree, quantification of BCL-2 expression and morphometric measurements of the heart.

Function analysis showed an impairment in left ventricular function on control group, which validated our proposed model of dilated cardiomyopathy. Furthermore, control group hearts had an enlarged left ventricle, with a thinner left ventricular wall and increased extracellular fibrosis. Although ACEi group had prevented the enlargement of the ventricular chamber and reduced fibrosis, left ventricular ejection fraction was not reverted when compared to control group. Yet, BS group prevented the decay of left ventricular function, on top of the diminished fibrosis and decrease in ventricular dilation.

Further analyses are being performed, and an article will be written and published.

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