Effects of bet-blocker therapy on the myocardial sympathetic Innervation in patients heart failure with preserved ejection fraction

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Abstract:

Objectives: Heart Failure (HF) with preserved ejection fraction (EF over 50%) is considered to be the most prevalent HF presentation. The autonomic nervous system (ANS) plays a key role in cardiovascular physiology and its dysfunction has pathophysiologic and prognostic implication in HF. 123 I-MIBG scintigraphy is and established method for evaluation of the functional status of adrenergic innervation in HF patients. Nebivolol is a beta-1-selective blocker with vasodilating properties related to nitric oxide (NO) modulation and there is now evidence that NO can modulate the beta-adrenergic stimulation in the normal human myocardium and with dysfunction. This study aimed to evaluate the integrity of the innervation (HFPEF) and the impact of nebivolol therapy in the sympathetic function.

Methods: It was a longitudinal study which included 24 consecutive ambulatory patients (60.6±10.9 years; 71% female) admitted in a heart failure clinic with a diagnosis of HFPEF. All patients were submitted to scintigraphy with 123 IMIBG and Minnesota Living with Heart Failure Questionnaire (MLHFQ) before and after they have received nebivolol 5 mg twice a day for a period of three months. HMR values standardized to low-energy collimation were used to define the H/M ratios and washout rate.

Results: There were no significant differences in H/M early (p=0.418), H/M delayed (p=0.532) and washout rate (p=0.644) in the basal evaluation compared to that obtained after three months of nebivolol therapy. Patients had significant improvement in clinical symptoms and quality of life as detected by MLHFQ changes after 3 months of nebivolol therapy (43.2 vs. 22.4; p < 0,01).

Conclusion: It was concluded that the use of nebivolol for three months does not improve cardiac sympathetic activity in patients with HFPEF in spite of impressive changes in quality of life. These data suggest that nebivolol may improve symptoms in HFPEF patients by additional mechanisms other than cardiac autonomic innervation regulation