FAVORABLE EFFECTS OF IVABRADINE ON SYMPATHETIC OVERDRIVE AND ARTERIAL STIFFENING IN HYPERTENSIVE PATIENTS WITH METABOLIC SYNDROME

Poster Contributions
Poster Hall B1
Saturday, March 14, 2015, 10:00 a.m.-10:45 a.m.

Session Title: Medical Management of Hypertension
Abstract Category: 22. Prevention: Hypertension
Presentation Number: 1108-133

Authors: Konstantinos P. Tsioufis, Kyriakos Dimitriadis, Evaggelia Koutra, Theodoros Kalos, Ioannis Liatakis, E. Andrikou, Panagiotis Vasileiou, Ioannis Andrikou, Dimitris Tousoulis, First Cardiology Clinic, University of Athens, Hippokration Hospital, Athens, Greece

Background: Hypertension and metabolic syndrome are related to sympathetic overdrive and arterial stiffening, while there are no data whether ivabradine modulates sympathetic activity and vascular abnormalities in this setting. The aim of this study was to assess the effect of ivabradine on muscle sympathetic nerve activity (MSNA) and arterial stiffness in hypertensive patients with metabolic syndrome.

Methods: We studied 36 patients with essential hypertension [age: 56±10 years, 30 males, office blood pressure (BP): 148/92±14/11 mmHg] on antihypertensive therapy with a fixed combination of perindopril/amlodipine. Patients were randomized with a ratio 2:1 to ivabradine (5 mg twice daily) or no ivabradine (control group). Metabolic syndrome was defined according to the Adult Treatment Panel III criteria. In all participants at baseline and at 6 months follow-up arterial stiffness was evaluated on the basis of carotid to femoral pulse wave velocity (PWV) while sympathetic drive was assessed by MSNA estimations based on established methodology (microneurography).

Results: Patients on ivabradine (n=24) compared to controls (n=12) did not differ regarding baseline BP, creatinine, glucose and lipid profile (p=NS or all). There was no significant difference in the reduction of office BP between the two study groups (p=NS). However, hypertensive patients in the ivabradine group were characterized by a reduction in carotid to femoral PWV from 11.5±0.9 m/sec to 9.8±1.2 m/sec (p<0.001) and sympathetic nerve traffic as reflected by MSNA levels from 86.2±2.5 bursts per 100 heart beats to 74.8±2.4 bursts per 100 heart beats (p<0.001) at 6 months. No significant changes in PWV and MSNA were observed in the control group (p=NS).

Conclusion: In hypertensive patients with metabolic syndrome, treatment with ivabradine reduces sympathetic activation and arterial stiffening as reflected by lower MSNA and PWV levels at 6 months follow-up. These findings suggest that ivabradine could exhibit additional therapeutic properties in the setting of dysmetabolic hypertension.