Abstract: P280

Lung endothelial cell dysfunction and pulmonary hemodynamics: is there any relationship?

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Introduction. Impaired lung uptake of 123I-metaiodobenzylguanidine (123I-MIBG) as a sign of endothelial dysfunction has been recently described in pulmonary arterial hypertension (PAH). Aim of this study was to confirm these findings and to compare PAH with dilated cardiomyopathy (DCM).

Methods. We investigated lung and heart 123I-MIBG kinetics in 13 consecutive patients with newly-diagnosed PAH and 11 DCM. All patients underwent right heart catheterization. Early and delayed uptake ratios of lung-to-mediastinum (L/M) and heart-to-mediastinum (H/M) were calculated on anterior planar images after intravenous injection of 123I-MIBG.

Results. The mean pulmonary artery pressure (PAPm) was not significantly different in PAH and DCM (37.8±11.8 vs 39±5.3 mmHg, respectively), in contrast to pulmonary vascular resistance (6.1±3 vs 1.3±0.9 Wood Unit, p=0.03) and pulmonary artery wedge pressure (7.85±2.64 vs 30±5.3 mmHg, p<0.001). Early and delayed L/M ratios in PAH were significantly lower than those of DCM (1.47±0.14 vs 1.98±0.11 and 1.40±0.13 vs 1.83±0.09; p<0.001), without a significant difference in pulmonary washout rate (WR, 23.9±5.5% vs 25.7±4.3%). There was no correlation between lung uptake and pulmonary hemodynamic parameters. Early and delayed H/M was reduced but not significantly different in PAH and DCM (1.73±0.20 vs 1.65±0.18 and 1.73±0.27 vs 1.58±0.19), as well as heart WR (22.7±6.1% vs 24.4±3.9%). A significant correlation was observed between delayed H/M ratio and PAPm (R=-0.61; p=0.03).

Conclusion: Lung uptake of 123I-MIBG is impaired in PAH and is lower than in DCM. Endothelial injury appears to occur regardless of pulmonary hemodynamics, contrary to myocardial sympathetic innervation, which is inversely related to PAPm.